MEDICAL TIMES

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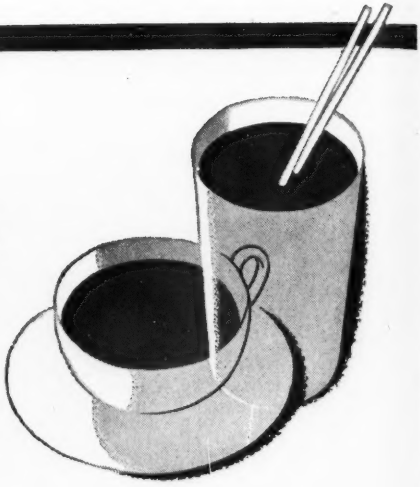
good taste in vitamins



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EDITORIALS

Dogberry, M.D.

A September news release from a university in the Middle West announced a promotion in the college of medicine to the rank of *distinguished professor* and stated that this rank had been held heretofore by only one other staff member; all of which, to us, has a flavor of incredible snobbery.

By the same token all other members of the medical faculty of this university are *undistinguished*. We suggest that these undistinguished professors be further broken down somewhat in the following manner:

Inferior Professor of Medicine
Mediocre Professor of Surgery
Commonplace Professor of Anatomy
So So Professor of Obstetrics
Déclassé Professor of Gynecology
Obscure Professor of Pediatrics
Inglorious Professor of Pathology
Unrenowned Professor of Bacteriology
Inconspicuous Professor of Otolaryngology
Second-rate Professor of Ophthalmology
Ordinary Professor of Public Health
Unknown Professor of Neurology

Aspiration of the Right Heart

Recent advances in cardiology, as for example Forssmann's and Cournand and Ranges' catheterization of the right auricle, have recalled to our mind a pioneer in this field—Benjamin F. Westbrook of Brooklyn, who in 1882 directly aspirated the right auricle as a means of relieving intense pulmonary congestion [*Medical Record* 26:705 (Dec. 23) 1882].

Westbrook passed his Codman aspirating needle, which was "about twice the caliber of an ordinary hypodermic needle," through the third intercostal space, close to the edge of the sternum, to the depth of about five centimeters, "when it communicated to the hand the sensation of being in the auricle . . . with the addition of the motion of the auricle, which causes the needle to move slightly to and fro with the pulsations of the heart."

This early step links with the modern technics.

Re-Examination of Sacred Premises

There is a small school of psychiatrists which denies the contention of their colleagues that there has been a decline in the intelligence of Americans. The position of this dissenting school is that no decline has been possible since the grade of intelligence has always been too low to admit of any decline.

This position finds something of a parallel in the claim now made by another small school to the effect that there has been no change in dental disease since recent studies of our Colonial forebears show that despite the absence of refined flour and sugar in their diet they had very poor teeth, contrary to our prevailing belief. The main point of this school is that something other than the lack of the coarse natural diet of our ancestors is responsible for our own widespread dental disease; it would seem not to be a consequence of faulty nutrition, as is so commonly believed.

Free Choice of Hospital by the Physician

Dr. Herman Goodman points out that the linkage of the qualified doctor of medicine and free choice of hospital in all the public and private plans for insurance medicine, in operation or proposed, is only *implied* and is largely an empty phrase. If the doctor has no hospital connection the plans really take no account of him no matter how well qualified he may be. In

—Concluded on page 334

Radiation Treatment of Infected or Hypertrophied Lymphoid Tissue in the Throat and Nasopharynx

John H. Vaughan, M.D.

Amarillo, Texas

This discussion is a follow-up of a paper, "Radium Treatment of Infected Tonsils," which I presented at the Physical Therapy meeting in St. Louis in September, 1930 and which was published in the *Archives of Physical Therapy* in January, 1931.

My decision to begin treating infected lymphoid tissue by irradiation was a result of reading some very interesting articles written in 1920 by Dr. Sanford Withers of Denver and also discussing the subject in 1926 with Dr. William Cameron of New York. One of my first treatments was on my own throat. Twenty years after having a clean tonsillectomy, my throat became infected and the whole of the Waldeyer's ring of lymphoid tissue became involved. It was infected for about 18 months and sore practically continuously. A week after treatment with a large dose of radium embedded in wax on my throat the infection left and has been well since then.

When I wrote the above article we had treated about 600 cases of infected throats with radium. At that time we applied the radium by embedding it in wax back of the angle of the jaw about ¾ inch from the skin. In 1937, we began treating these cases with x-ray and now we use it in practically all cases. The reason we prefer to use x-ray rather than radium is that the x-ray gives a uniform dose over the entire Waldeyer's ring. We treat both sides of the throat, giving a dose once a week for 5 weeks. If at the end of 2 months the results are not satisfactory we give 2 more treatments. We also inform those patients having tonsils that we will remove the tonsils surgically, without cost to them, if they are dissatisfied with the results of the x-ray treatments. We do this to encourage them to return for observation.

We have now treated about 2,500 cases of which the tonsils had been surgically removed prior to treatment in approximately 35 per cent of the cases. In the past year we have treated 130 cases of infected throats. In 49 of the cases the tonsils and adenoids had been previously removed surgically. Most of these 49 cases were referred to us by eye, ear, nose and throat specialists.

We use the following factors in these treatments: 200 Kv; 15 Ma 0.5 cu—1A1—50 Cm. Distance. The entire head is covered with a piece of lead ⅛ inch thick which has a hole in it that is large enough for the rays to penetrate only the Waldeyer's lymphatic ring. If the throat contains massive adenoids it is advisable, though not necessary, to remove them surgically prior to treatment.

Since we all know that each throat we treat contains some infected or hypertrophied lymphoid tissue and that lymphoid tissue is very sensitive to radiation, we should get marked improvement in each case. In our opinion, the only infected tonsils which we cannot cure with irradiation are those which contain closed crypts or have a walled-off abscess.

Summary: As a result of our treating throats over a period of 21 years and my observation of the excellent results attained by other radiologists in this work, it is my opinion that in the near future at least 90 per cent of the infected throats will be treated by radiation. Since radiation is in no way harmful, if properly given, and the patient is not subjected to an anesthesia, pain or danger of hemorrhage and trauma, the use of this form of treatment should continue to increase at a rapid rate.

806 Fisk Building.

Recognition of Non-opaque Foreign Bodies in the Tracheobronchial Tree

Report of One Case

Henry B. Smith, M.D., F.A.C.S., and Percival A. Robin, M.D., F.A.C.R.
Hempstead, New York

Non-opaque foreign bodies are relatively common in the bronchi. Many signs and symptoms contribute to the recognition of this entity. A definite history of inhalation or ingestion of a foreign body may or may not be obtained. If present, it is usually associated with pain, choking, brief or persistent coughing, and may be followed by wheezing. These are findings that contribute to a definite diagnosis. Later, symptoms may include air hunger, persistent cough, wheezing, and a low-grade elevation of temperature.

On examination the patient may exhibit a difference in expansion of the two sides of the chest; diminished breath sounds over one area; poor aeration; and retention of secretion. A wheezing sound due to the air passing by the foreign body is often heard.

The radiographic examination is essential, and helps to confirm the presence of a non-opaque foreign body by indirect signs. As a rule, there is only a partial stenosis produced by the foreign body and associated edema of the adjacent mucosa of the bronchus. This permits a ball-valve type of obstruction. The signs of obstructive emphysema can be recognized roentgenoscopically, or on the roentgenogram. The radiographic examination, made with special technic, shows the following:

A roentgenogram made at the end of deep inspiration shows both sides of the chest to be equally aerated. A film taken at the end of expiration, however, shows that the lung on the affected side remains in varying degrees of expansion, whereas on the non-affected side there is a normal collapse of the lung tissue at the end of expiration. In marked obstructive emphysema, the mediastinum is shifted away from the affected side in contradistinction to true atelectasis, where there is a shift to the involved side. On fluoroscopic observation, a swinging motion of the mediastinum is noted during inspiration and expiration. These signs are not present when complete stenosis of the bronchus occurs, for then the characteristic roentgen manifestations of atelectasis are noted.

An unusually definite picture of obstructive emphysema was recently observed in a child. L. M., aged 14 months, had a mild upper respiratory infection and received some cough medicine for a period of forty-eight hours. On Christmas day, 1946, the patient was given one-half of a peeled apple from which the core had not been removed. At that time she had a coughing spell, and was picked up, coughing severely. She vomited small pieces of apple, and then seemed to be better. However, wheeze persisted, and the local medical doctor was called. He felt that the wheezing might be due to a foreign body. Because of the acute upper respiratory infection, it was decided that observation of the patient for a few days should be carried on without any definitive treatment. Although the child improved, a slight wheeze persisted.

Fig. 1. A roentgenogram of the chest in deep inspiration. There is very slight generalized increase in density over the right lung field.

On January 15th, 1947, a radiographic examination was made. The findings were as follows:

Films were made at the end of both inspiration and expiration. (Fig's. 1 and 2). They show definite evidence of obstructive emphysema. This is characterized on the expiration film by relative over-aeration of the left lung field, when it is compared with the right. Also, there is a characteristic swing of the heart, mediastinum and trachea to the right on this film. On the film made at the end of inspiration, there is only a slight increase in density over the right lung field. The findings are typical of a non-opaque foreign body probably obstructing the main bronchus on the left side. The heart is normal in size and configuration.

After a definite diagnosis had been made through radiographic and clinical findings, a bronchoscopic examination was decided upon. On examination, a small piece of the carpel, i.e., (leathery inclosure of the apple seed), was removed from the left main bronchus. Inspection of the bronchus showed that it was partially obstructed by severe edema of the mucous membrane.

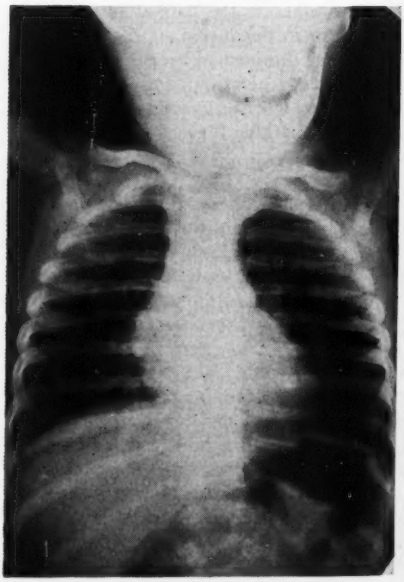
A follow-up radiographic examination on January 27th showed the following:

Both lungs are equally radiable, and there is no shift of the heart or mediastinum to the right. The findings indicate that the obstructive emphysema has cleared up after the removal of the non-opaque foreign body.

Clinically the patient has completely recovered.

Conclusion—Because of the definiteness of the roentgen findings in a case of obstructive emphysema, a diagnosis of a non-opaque foreign body could be made. In addition, the bronchoscopist had the advantage of localization of the foreign body within the bronchial tree. This saved unnecessary investigation. Upon removal of the foreign body the symptoms which caused concern subsided rapidly.

Fig. 2. A roentgenogram of the chest taken at the end of expiration. Note the relative increase in density on the right and the marked shift of the heart, mediastinum and trachea to the right.



CANCER

Serial Surveys of Current Concepts and Activities

Department Edited by
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Carcinoma of the Salivary Glands

PAROTIDECTOMY: INDICATIONS AND RESULTS. Hamilton Bailey. *Brit. Med. Jour.,* 1:404 (March 29) 1947.

The author says: "As an experienced teacher I am convinced that in the whole realm of clinical surgery there is not a condition that approaches mixed parotid tumours for misconceptions. Here is a neoplasm that remains supremely curable for years, sometimes for a decade or more. Yet, because of misconceptions concerning its behaviour and its treatment, it is allowed to pass from curability to incurable malignancy. Worse still, while it is as yet small and encapsulated, malignant cells, previously locked up by Nature, are let loose by surgical interference."

In relation to the operative technique Bailey says that no attempt should be made to remove any parotid tumor until the whole gland is in full view. Only then is it possible to tell how deeply the tumor "plunges into the gland." Ligation of the external carotid artery will reduce operational hemorrhage. At all operations on parotid tumors the wound should be kept moist with 60.0 per cent alcohol. In Bailey's experience with thirty-three total or subtotal parotidectomies and about an equal number of extracapsular resections, there was but one fistula. This fistula healed within a year and has remained healed.

In 1934 Stein and Geschickter (6) reported the records of 241 tumors of the parotid gland from a total of 50,000 surgical patients. Of these 241, forty-two or 17.4 per cent were malignant and 199 or 82.5 per cent were benign. They believed at that time that both surgery and irradiation have their place in the treatment of these tumors. The benign tumors are best treated with complete excision, being careful to avoid injury to the facial nerve. The malignant tumors should be irradiated first and excised later if they become freely movable. If they do not become freely movable, irradiation should be continued. If they become removable, interstitial irradiation should be applied to the tumor bed. Benign tumors are usually circumscribed, firm, and rubbery, and microscopically show a large amount of connective tissue and myxomatous stroma. The malignant tumors are adherent and may be hard or soft. They grow rapidly and, microscopically, present adenocystic basal cell characteristics.

In the same year Martin and Elkin (2) reported twenty-four cases of salivary gland tumor. They recommended early removal of the primary growth followed by radium and roentgen irradiation, which offer the greatest hope for cure. The prognosis is not good. Following the removal of the original tumor, recurrences are likely to be more malignant.

In 1935 Calvin Stewart (7) after a study of thirty-nine cases drew the following conclusions: (1) All tumors of the parotid region should be considered to be malignant until proved benign by microscopic study; (2) surgery is the principal method of treatment; (3) ligation of the

external carotid artery is a safety method to be used at the beginning of an operation in the parotid region; (4) preoperative roentgen irradiation and radium implants are to be considered; (5) occasionally radium implants have successfully controlled recurrences.

In 1939 MacFee (1) reported the results of the study of twenty-seven cases of salivary gland tumors from St. Luke's Hospital (New York, N. Y.) and the New York Hospital (New York, N. Y.). Of the twenty-seven growths twenty-two were in the parotid gland, four in the submaxillary and one in the sublingual gland. He says, "Some of the tumors are malignant from the beginning; others appear to result from malignant changes in primarily benign tumors." Metastasis tothe regional lymphnodes, according to MacFee, is relatively infrequent; in the present series of cases approximately 15.0 per cent, actually four, of the twenty-seven cases (14.8 per cent). Metastasis to the lungs is more common than is generally recognized. In this series of twenty-seven cases, eleven had roentgenologic chest studies with evidences of pulmonary metastasis in eight (72.7 per cent of those examined). MacFee recommends routine chest roentgenogram in such cases. Surgery and postoperative irradiation is the method of choice in treatment. Of the twenty-seven cases, five of the patients survived for more than five years (18.8 per cent) but two of these patients had metastases. On the other hand, five patients died of recurrence from six and one-half to twenty-two years after the first appearance of the tumor. The prognosis, consequently, is not very good, and the author thought not more than 25.0 per cent of five-year survivals can be expected. There is an excellent bibliography.

In 1939 Morris Smith (5) reported the case of a woman, aged 31 years, who in 1924 was operated upon for the removal of a tumor of the floor of the mouth of three years' duration. Under the impression that the lesion was inflammatory, it had been incised on several occasions. On removal, histological study indicated that it was a "mixed tumor." Four years later there was a recurrence, and a second recurrence a year after the removal of the first. Three years later (1932) there was a third recurrence which was treated with irradiation. In 1937 lung metastases were demonstrated by roentgenogram.

Between July 1, 1912, and July 1, 1936, 65,351 patients were admitted to the Barnard Free Skin and Cancer Hospital (St. Louis, Mo.). Seventy of these patients had tumors of the parotid gland (0.1 per cent). Thomas Martin (3) analyzed these cases. Of the seventy, thirty-four were diagnosticated mixed tumors and thirty-six malignant tumors. In eighteen of the mixed tumor cases the diagnosis was confirmed by histological study. In fourteen of these, the tumor mass was removed surgically, and in ten the patients showed no evidence of recurrence for periods varying from six weeks to nine years. Of the thirty-six malignant tumors, seventeen were treated surgically. Six of these showed no recurrence in from seven weeks to fifty-seven months. The author says that irradiation "except for palliation has not been effective."

In 1945 Stewart, Foote and Becker (8) reviewed the histology of about 700 major and minor salivary gland tumors of all types seen in Memorial Hospital (New York, N. Y.) between 1928 and 1943. They found forty-five growths to which the term muco-epidermoid is believed to be applicable. Such growths, they believe, originate from the mucous cells that are to be found, after careful search, in the duct epithelium of both the major and the minor glands. It was found that these growths may be divided into benign and malignant varieties. But it was pointed out thit it might be better to call them a "relatively favorable" group and a "highly unfavorable" group, the existence of metastasis being the characteristic feature of the malignant tumors. Thus far the authors have found no metastases from the tumors called "benign." In the group called malignant the histologic structure is like that of growths which have the "ability to produce regional node and distant metastases." Twenty-six of the growths were considered to belong to the "benign

type" (57.7 per cent) and nineteen were considered to belong to the malignant type (42.2 per cent). In some of the "benign" tumors, "about a third," areas were found composed of cells that "appeared hydropic and swollen." In general the cells of the benign tumors vary over a very wide range of structural variation and there are other modifications due to "overproduction of mucus, distension of canalicular components, secondary interstitial inflammation and spontaneous necrosis." The outstanding feature of the malignant tumors is "diffuse proliferation of rather small, moderately hyperchromatic, rounded and oval cells in sheet-like arrangement, with tendency to palisading of the outer layer of cells which surround the proliferating sheets and pegs of tumor."

There are two illustrations of areas from the same tumor, one of which shows "malignant transformation of an alveolar ridge tumor" which had been present twenty-five years. The other shows an area of papillary cyst adenoma, benign. (Figures 13 and 14.)

The variable structure of the primary tumors is sharply reflected in the metastases. In four instances it was possible to demonstrate mucous cells in large numbers in the metastatic nodules. There is a tendency for these growths to occur in young people. Fifteen of the benign tumors were first noticed by the patients when they were less than forty years of age and six of the metastasizing tumors were found in patients under forty years of age. The malignant tumors are highly fatal. The benign tumors (original diagnosis) tend to recur, and of the twenty-six patients in the "benign" group, only nine were known to be living and free from disease at the end of the five-year period. Two of these were treated with irradiation alone; three were treated with surgical excision alone; four were treated with combined irradiation and surgery. Of the nineteen in the malignant group, only one patient is alive and free from evidence of tumor.

Between 1928 and 1936, inclusive, at the Mayo Clinic (Rochester, Minn.), there were 210 consecutive cases of primary parotid neoplasms which had been removed surgically and which were sufficient numerically, according to Quattlebaum, Dockerty and Mayo (4), to warrant conclusions concerning the incidence of parotid carcinomata of the cylindroma type and concerning the results of a follow-up program. Of these 210 cases, twenty-one seemed to warrant a revised diagnosis of "adenocarcinoma of the cylindroma type" (10.0 per cent). The histological characteristics are to be found in a previous contribution by Dockerty and Mayo, (*Surg., Gyn. and Obstet.*, 74:1033 (June) 1942).

The cases of this type of parotid tumor are characterized by the presence, clinically, of sharp, radiating pain and local fixation of the tumor. In other forms of parotid tumor pain is "an unusual feature" and "its presence . . . should make one alert to a possible underlying adenocarcinoma of the cylindroma type." "Partial or complete paralysis of the facial nerve was a common finding." Surgically, the growths should be widely excised because they are usually infiltrating and without capsule. Twenty of the twenty-one patients have been followed for five years or more; eight died within that period. Two are dead in more than five years; one in thirteen years and the other in six and one-half years. Four are living with repeated recurrences. One was living for thirteen years and then was lost. Five are known to be living without recurrence, seven to fourteen years. Metastasis, five to the lungs, was the cause of death in eight cases. The authors conclude that "solid tumors of the parotid gland should be assumed to be malignant until proved otherwise." And they recommend more radical surgical procedures if better results from treatment are expected.

It seems to us that the opinions of the majority of the students of this problem may be summed up in the first quotation from the paper by Dr. Hamilton Bailey. It seems unwise to leave in the body of

—Concluded on page 334

The Complex Problem of Rheumatic Fever, Its Complications and Treatment

This summarization attempts to cover all of the known therapeutic information on the subject and is designed as a time-saving refresher for the busy practitioner.

Rheumatic fever annually kills more persons under twenty years of age than tuberculosis, epidemic meningitis, measles, diphtheria, scarlet fever and infantile paralysis combined. In the first two decades of life rheumatic fever causes more deaths than all of the communicable diseases plus pneumonia.[7]

It is a common belief that rheumatic fever is primarily a disease of children. This is not true for adults are stricken in great numbers. During the war 40,000 in the armed forces alone were victims. A study of the young men rejected because of heart disease for military service in the five largest cities of the United States revealed that rheumatic fever was the cause in the majority of cases.[2]

The severe types of rheumatic fever occur more frequently in the temperate zone but the disease is not limited to this zone as was thought in the past. Sampson[3] and his colleagues have shown that it is almost as prevalent in subtropical climates. Huntington[4] reports that the acute fulminating type is often seen in the tropics.

Examination of the literature indicates a variance of opinion among authorities on certain phases of rheumatic fever. Consequently this article attempts only to summarize *all* methods of drug therapy.

Definition

Rheumatic fever is also known as acute rheumatic fever or inflammatory rheumatism. The choice of the adjective rheumatic is somewhat unfortunate since this disease may exist without any of the bone and joint symptoms characteristic of rheumatism. These symptoms may exist to some slight degree but in sufficient intensity to cause cardiac damage. A general description of this disease is as follows: Rheumatic fever is a systemic disease occurring most frequently among children. It is an acute, non-contagious disease. It is characterized by a polyarthritis and a marked selective action on connective tissue, serous membranes, and vascular structures.[5] It is a systemic nonsuppurative inflammatory disease which manifests itself in many shapes and in varying severity and duration. Observation has shown that the inflammation is a hypersensitization phenomenon and extends to all of the bodily structures from the skin to the smallest subdivision of the viscera.[1]

Etiology

The theory has been advanced that rheumatic fever is hereditary and is a Mendelian recessive character.[6] This is not definitely established although it cannot be completely ignored for it is true that children born to parents who have had rheumatic fever are more susceptible to this disease. This is especially evident in those children suffering from an infection caused by the hemolytic streptococcus. Among these are included tonsillitis, scarlet fever and otitis media.[7,8]

Environment

Environment appears to be more important than heredity. Rheumatic fever is more prevalent where living conditions are overcrowded and where there is a lack of sunlight and where excessive dampness is present. This was the experience of the Army and Navy in their training centers. Certain nutritional factors serve to protect the person from such etiologic agents as the beta-hemolytic streptococcus. This is probably true only in the sense that good nutritive status seems to increase resistance to infection in general.

From the Editorial Research Department of the MEDICAL TIMES.

Prevention

In the prevention of rheumatic fever all infections, even minor ones of the upper respiratory tract, should be avoided. It is most probable the inciting agents in so-called rheumatic fever epidemics may be upper respiratory infections of streptococcic origin which act as catalysts. The fact that rheumatic fever occurs most often in late winter at about the same time as such minor respiratory infections as sore throat and the common cold prevail leads one to believe that it is more than coincidental.

Since nothing can be done about heredity there are certain other factors which can be carefully controlled, which should be done in those cases where there is an hereditary background of rheumatic fever. The conditions under which the child works and plays should be observed. His food should be wholesome and nutritious. He should be carefully guarded against exposure to respiratory infections. The condition of his tonsils should be known and any infected dental socket immediately dealt with. Of course it is necessary to stress that all of this be done without disastrous psychological effects.

Phases of Rheumatic Fever

There are four phases of rheumatic fever.[1] The illness during which the individual first shows evidence of some infection caused by the beta-hemolytic streptococcus is the first phase. Every person with a throat infection caused by this organism should be kept under continued observation for rheumatic fever for five per cent of those trainees in the armed forces who developed a suppurative hemolytic streptococcic infection in the upper respiratory tract also developed rheumatic fever signs and symptoms. The infection with this organism may manifest itself as pharyngitis, tonsillitis, otitis media or a sore throat with a scarlatiniform rash.

Latency Period

Following the first phase is a period of latency lasting from one to four weeks. During this time the symptoms noted are not limited to rheumatic fever but may be observed in any number of conditions. The patient is generally run-down or debilitated; tires easily; weight falls below normal average; appetite is poor and aversions to certain types of foods may occur; may have a mild fever; and pulse and heart action may be more rapid. Nervous instability may develop and a pallor of the skin be noted. Laboratory tests show a persistent leukocytosis, an accelerated blood sedimentation rate and a gradual increase in the level of the antistreptolysin titer.[9] This period of latency is difficult to determine and the physician should not overlook it.

Active Stage

The active stage of rheumatic fever is the third phase.[1] This may begin very abruptly and severely or it may be insidious and difficult to detect for some time. The severity of the onset usually varies with the age of the patient. Generally the younger the patient, the more severe is the onset. An acute illness follows the latent period. This is characterized by inflammation moving from one joint to another, beginning in the larger ones such as the knees, ankles and so forth, and at times even involving hip and shoulder joints. The heart action is more rapid than it should be to correspond with the rise in temperature which occurs. There also occurs a loss of appetite. Severe drenching sweats alternate with periods of hot and dry skin. The tongue and lips are dry and the face is flushed. Although the patient is nervous and restless it is difficult to move due to the pain in the joints. The tissues around the joints swell and are hot and sensitive to the touch. The more active the patient has been the more severe are the manifestations of the disease in the joints.

The fever if present may be either moderate or severe and lasts about ten to eighteen days after which it gradually subsides. A low degree of fever may continue for several months. The rapid heart action usually occurs with or without fever and remains as long as there is any activity. Carditis is often manifested early in the third phase but most frequently occurs later. Pneumonitis, as evidenced by short-

ness of breath, tachycardia, fever, pleuritic pain, malaise and cough, occurs in many patients, particularly when the condition exists for a long period of time.[10] Return of appetite and a gain in weight are good evidence of a subsidence in the condition.

Abdominal Pains and Some Other Phenomena

Frequent and recurring abdominal pains due to peritoneal inflammation often occur and should not be confused with acute appendicitis. Nervous twitching may occur in young patients but is not so likely to in older groups. This also applies to the occurrence of small round nodules, visible under the skin. They are movable, nontender, variable in size and consistency and usually appear in groups over the bony prominences and sometimes on the tendons. Children may experience nosebleeds of short duration due to an increase in capillary permeability and fragility.[11] This is not quite so frequently seen in adults.

Criteria for Diagnosis

The criteria for the diagnosis of rheumatic fever thus have been divided into two groups. The five major ones include a previous history of a rheumatic fever attack, carditis, fever, migrating polyarthritis and chorea. Early recognition of chorea is an aid in diagnosis. The minor criteria are eight in number and include in order of importance: pneumonitis, tachycardia, subcutaneous nodules, erythemas, epistaxis and purpura, loss of weight and abdominal pain.[12,13] Another subdivision places the subcutaneous nodules in the major group and fever in the minor group and includes abnormal laboratory findings in the latter group as well.[14]

Quiescent Period

Following the third phase of rheumatic fever activity is the fourth phase of inactivity in which the disease is in a period of quiescence.[1] This period continues until the rheumatic state is again reactivated and may be either of long or short duration. Reactivation of the rheumatic state is considered in effect only when the major and minor diagnostic symptoms again prevail. Many cases which have been considered as reactivated actually are not, for fatigue or exposure will cause mild pains due to muscular stiffness and soreness of the joints.

Relapsing Disease

Essentially rheumatic fever is a chronic relapsing disease. The four phases which form its course usually cover a period of years. Recovery from rheumatic fever is a common occurrence and every individual has a fighting chance. If children who have this condition survive puberty they are usually free of symptoms in adolescence and young adult life. In most cases the heart is usually involved but the damage is very often repaired to such an extent that the individual can lead a normal adult life. A great deal depends upon the severity of the infection at onset. The younger the age and the more severe the infection the less chance the child has of survival.

A study of over 10,000 rheumatic fever patients of military age[1] revealed only 13 deaths from a primary attack of rheumatic fever. The causes of death in this group in order of frequency were: congestive heart failure, pneumonitis, coronary occlusion with myocardial infarction, and shock resulting from necrosis of the adrenal gland.

Differentiation

In diagnosing rheumatic fever it is necessary to follow closely the criteria mentioned above and the clinical course of the disease for there are many syndromes in which the symptoms are similar. Among these are acute osteomyelitis, undulant fever, rheumatoid arthritis, chronic osteoarthritis, tuberculosis, Reiter's syndrome and the collagenous diseases. These can usually be ruled out by the presence of only one or two of the major and minor criteria previously enumerated.

Atypical Clinical Features

A study of young adults in a large naval hospital, where many patients with proved or suspected rheumatic fever were under observation, revealed that there are many atypical clinical features of rheumatic fever.[15] In this study the most valuable and

common diagnostic criteria were found to be arthralgia, carditis and particularly abnormalities in the electrocardiogram, fever and a persistent elevation in the sedimentation rate. The other major and minor criteria were not so common. Clinical and laboratory tests should be made if a patient, who has had a hemolytic streptococcic respiratory infection, shows continued fatigue, failure to convalesce satisfactorily, continual or reappearing low grade fever, or arthralgia. The most striking clinical features noted in these atypical cases were: minimal or complete absence of articular symptoms, nonarticular involvement, onset with major manifestations in the hands and feet, prodromal aching for days to weeks prior to the onset of acute arthritis, recurrent acute arthritis of short duration with apparently complete subsidence between attacks, absence of fever during the acute stage, onset with fever of obscure origin, association of rheumatic fever and Marie-Strümpell spondylitis, onset with pericarditis and pneumonitis and subclinical recrudescence.

Pathology

There are numerous pathologic findings in rheumatic fever.[15] Some are characteristic of the condition whereas others are not. However, they do indicate that tissue involvement is widespread.

Clinical Types

Rheumatic fever can be classified into four distinct clinical types.[1] The first and most serious one is the acute fulminating type which is usually found only in childhood. It is characterized by sudden onset and occurs in the patients most susceptible to the disease. The mortality rate is low in cases of primary attack but reactivation is more likely to occur. The second type or subacute polycyclic type occurs more frequently than the former but is less severe. Its characteristics, however, are very similar to the acute fulminating type. The most frequently observed type is the third, the subacute monocyclic. The onset is usually mild and scarcely recognizable. A fourth type known as the subclinical type is found in a small group of cases. In this group are included the individuals who are discovered to have signs of rheumatic heart disease when examined following an upper respiratory tract infection.

Therapy

The chief problem in the therapy of rheumatic fever is the prevention of permanent disabling damage to the heart. Since the etiology of the disease remains unknown, no effective methods have been found to destroy the causative agent, and no means have been developed to prevent the inflammation in the vascular tree. It is necessary to employ all available therapy to reduce the damage to the heart as much as possible and to prevent death. This could be avoided if it were possible to weaken and shorten the first two types, the acute fulminating and subacute polycyclic, and if the third and fourth types, subacute monocyclic and subclinical, could be prevented from causing such severe inflammatory-like reactions in the vascular tissues. Rheumatic fever therapy is divided into two phases as follows: treatment during the active stage and that administered during the quiescent state.[1]

Institution of Therapy

It is difficult to determine just when therapy of the first phase should begin. However, as mentioned previously, any patient recovering from an hemolytic streptococcic infection of the throat is susceptible to rheumatic fever and thus should be observed closely. If the individual is slow in recovering, tires easily, has a low grade fever in the evening it is possible that rheumatic fever may be developing. Laboratory tests are also of value. A high antistreptolysin titer is most important as evidence of a possible rheumatic fever condition. An accelerated blood sedimentation rate is also indicative of rheumatic fever. Early recognition of the active stage is imperative. A carefully planned and executed regimen of rest and therapy should be started at once.[1]

Rest, Environment, Nursing Care

Rest, with very limited activity in bed, is necessary and should be continued until

the condition shows signs of improving. At this point the degree of rest in bed can be relaxed gradually as the signs of activity of the disease disappear. The environment should be quiet and conducive to sleep. Diversions to ease the mind of the patient should be planned. The condition of the patient is improved considerably by intelligent and considerate care. The elementary nursing principles such as a daily bath with tepid water and clean linen should be observed. Careful attention should be paid to the diet and to elimination.

Diet

A highly nutritious diet of at least 3500 calories daily should be given. The form of the diet, whether it be liquid, soft or solid, is dependent upon the temperature and general condition of the patient.[1]

It is not necessary to use supplementary vitamins as a routine but a varied diet should be given. However, in some cases where the illness has been very severe and feeding difficult, vitamins are of value. It was once thought that administration of ascorbic acid brought about a more rapid convalescence but this has not been proven to be so. To some extent it does decrease the gastric irritation which attends the administration of salicylates. Capillary fragility as evidenced by petechial hemorrhages is common to the rheumatic fever state. Vitamin P or hesperidin has shown no value in lessening the hemorrhages; neither has vitamin K nor calcium in large doses.[11] Anemias sometimes occur as secondary conditions during the active rheumatic state. Iron, in the form of ferrous sulfate, may be given in doses of 0.4 Gm. (6 grains) three times daily. Chorea has been controlled by a high ketogenic diet and high salicylate blood level.[1]

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—To be continued

The Evolution of Inguinal Hernia Surgery

VIII

Alexander Hugh Ferguson, Professor of Surgery, Chicago Postgraduate Medical School, described his operation for the radical cure of hernia in 1899 before the Section on Surgery and Anatomy of the American Medical Association (*J.A.M.A.* 33:6, July 1, 1899). His paper reported the results of sixty-four operations. At the time he presented the paper there had been no recurrences.

Ferguson believed that a deficient origin of the internal abdominal oblique and of the transversalis muscles at Poupart's ligament accounted for recurrences after operation for radical cure. What he found in fifty dissections confirmed his conviction. He adopted the semilunar incision and turning down of the flap over the thigh when studying the reasons for recurrences after the conventional techniques. The outcome of this careful work was the planning of the Ferguson operation described in this article, including the retention of the semilunar incision for thorough exposure of the factors at fault, which he claimed he was always able to find. In his paper he describes an angle between the lower border of the internal abdominal oblique muscle and the inner aspect of Poupart's ligament wholly unprotected by the internal oblique and transversalis muscles. This deficiency, he pointed out, accounts for the original hernia and for recurrence unless corrected.

The semilunar skin incision begins over Poupart's ligament, 1½ inches below the anterior superior spinous process of the ilium, extends inward and downward, circumscribing the internal abdominal ring, and terminates over the conjoined tendon near the pubic bone. The external abdominal ring and intercolumnar fascia are cut through and the fibers of the external oblique muscle separated directly over the inguinal canal, far beyond the internal ring, over the surface of the internal abdominal oblique muscle, and up under the skin, to a point nearly opposite the anterior superior spine of the ilium. The aponeurosis of the external oblique muscle is retracted to bring into sight the deep structures, viz., the contents of the inguinal canal, the whole sac with its adhesions, the spermatic cord, the ilio-inguinal nerve, the usually enlarged internal abdominal ring, perhaps an accumulation of subserous fat, the cremasteric muscle, conjoined tendon, internal oblique muscle and its deficient origin at Poupart's ligament, the transversalis fascia, and the internal surface of Poupart's ligament. The sac is emptied and then ligated high up over an inserted finger, cut off, and the stump dropped. The cord is never disturbed. The next step is to take up the slack in the transversalis fascia by means of interrupted or continuous sutures to make an accurately fitting ring for the cord. The internal abdominal oblique and transversalis muscles are now sutured to the internal aspect of Poupart's ligament to restore their normal origin. Finally, the separated edges of the aponeurosis of the external oblique muscle are brought together with a continuous suture and the external ring is restored.

A. C. J.

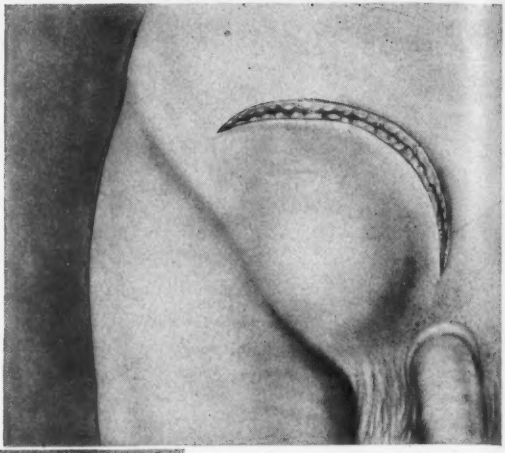

Fig. 1. The semilunar skin incision.

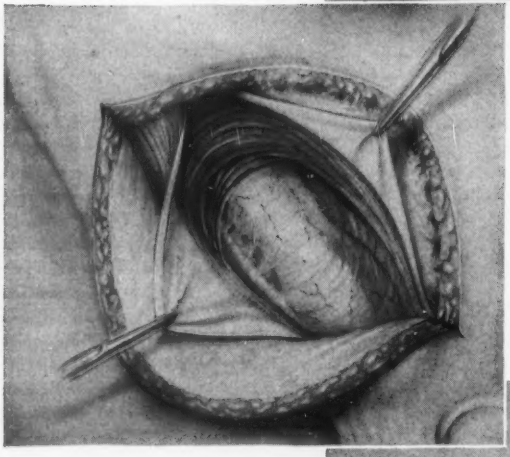

Fig. 2. After cutting of the external ring and separation of the fibers of the aponeurosis of the external oblique retraction of the aponeurosis enables one to view the deep structures.

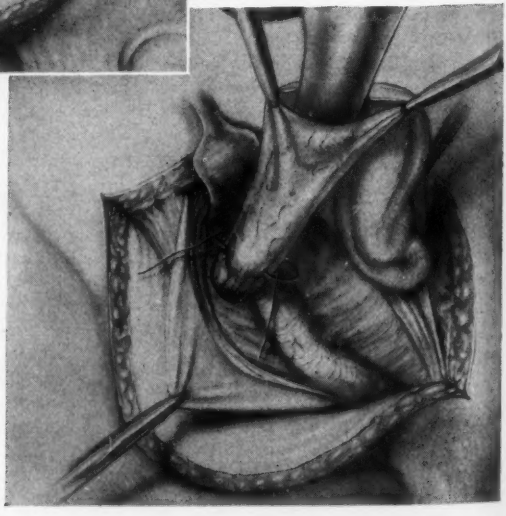

Fig. 3. Treatment of sac.

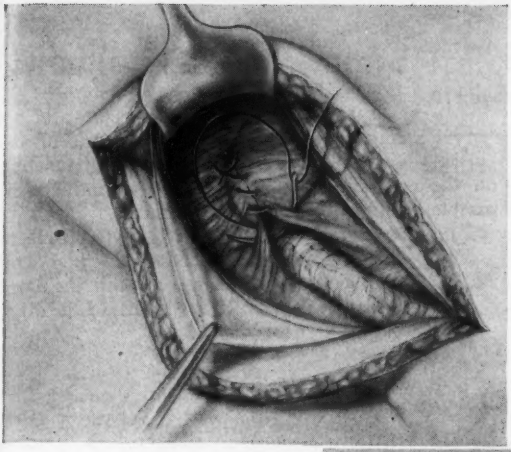

Fig. 4. Sutures take up the slack in the transversalis fascia and make a carefully fitted ring for the cord.

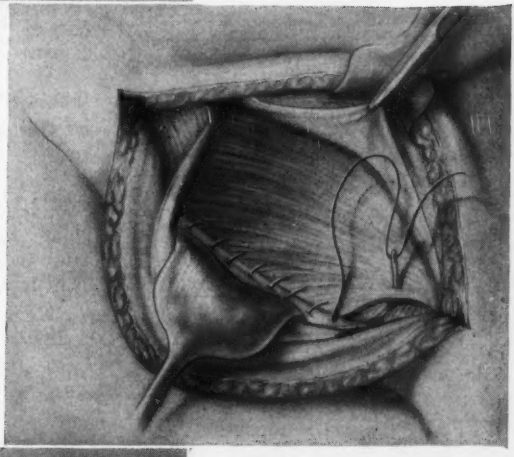

Fig. 5. Suturing of the internal oblique and transversalis to the internal aspect of Poupart's ligament restores their normal origin.

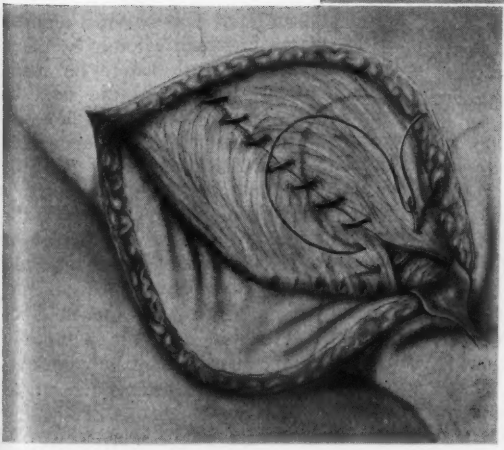

Fig. 6. A continuous suture brings together the separated edges of the aponeurosis of the external oblique and the external ring is restored.

EDITORIALS

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other words, at all points there is insistence upon free choice of doctor on the part of eligible patients but no comparable insistence upon free choice of hospital on the part of all qualified doctors. This limitation is true even of the A. M. A. plan. It is only at the ambulatory office level that the unattached doctor can function.

Lay participants in the plans are not told these things and an element of deception if not of fraud figures in the matter, yet Goodman seems to believe in the possibility of an honest adjustment.

Obviously, this subject relates itself to the problem of the closed shop and monopoly in medicine, already discussed in recent issues of the MEDICAL TIMES.

CANCER

—Concluded from page 325

a patient a growth which is capable of passing from benignancy to malignancy, hoping that the change would not take place.

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Gems from the Literature of Cancer

I

"To employ intensive irradiation of bone lesions on roentgenographic evidence alone is an unwarranted intellectual obeisance to a fallible diagnostic measure."

[Ian MacDonald and John W. Budd. *Surg., Gyn. and Obst.* 82:81 (Jan.) 1946]

A Rheumatic Fever Library

The Colorado Rheumatic Fever Library has been established at the University of Colorado School of Medicine in response to the great need for the assembling in one place of all that has been written on the subject of rheumatic fever. It is desired that this Library contain reprints or copies of every article that has been written on rheumatic fever. At first efforts will be made to collect all of the reprints that may be available, and when reprints can no longer be obtained, photostatic or typewritten copies of desired articles will be made. Publications, or copies thereof, from countries outside of the United States, will be collected in a similar manner and where necessary will be translated. This material will be cross-indexed and assembled in bound volumes by years. The collection will ultimately be made available to all workers in the field of rheumatic fever in the form of a photostat and an abstract service. It is hoped that this body of information, readily available to all interested persons, will form a major contribution to the solution of the basic problems of etiology and therapy of rheumatic fever. Valuable assistance can be given by all libraries, all medical journals, all those writing in the field of rheumatic fever and all those willing to dispose of reprint collections by contributing papers relating to rheumatic fever, as well as by making a practice of sending reprints of future publications.

Anemia, A Review of Its Causes and Therapy

Presented herein is a summary of the various types of anemia, symptoms and effective therapy designed as a time-saving refresher for the busy practitioner. Reprints available.

—continued from October issue

Cooley's Anemia

Cooley's or Mediterranean anemia appears to be an hereditary condition which sometimes occurs congenitally in children born of Mediterranean parents. It is caused by a deficiency in hematopoiesis for which the cause has not been determined. It usually appears in children between 6 months and 3 years of age. It may be fatal very early in the course of the disease or it may progress more slowly. Patients with a mild form may live to adulthood but usually die before reaching 10 years of age. There is no known effective therapy. The disease is characterized by a severe anemia. The spleen and liver are enlarged; the skin is yellow; and there is vomiting, anorexia and severe fatigue. The bone marrow is hyperactive and produces an abnormal red cell. The bone marrow in all bones widens and replaces the cortex. The hemoglobin is usually as low as 3 to 8 per cent and the red cell count is reduced in severe cases to as low as 1,000,000, the red cell volume is reduced, and the color index is usually below 1. The number of leukocytes is usually larger and the platelet count normal. Leukocytosis occurs accompanied by myelocytes and myeloblasts. The number of monocytes and lymphocytes may be greater. The red cells vary in both size and shape showing both anisocytosis and poikilocytosis. A greater majority of microcytes showing hemoglobin at the periphery are usually present. Target cells in which the hemoglobin is only in the center may also be observed. The fragility of the cells is not increased. There usually are present Howell-Jolly bodies, stippling and polychromatophilia. Howell-Jolly bodies are small, round or oval bodies, pinkish or bluish in color, which can be shown by staining with Giemsa's stain. They are believed to be fragments of broken up nuclei of nucleated erythrocytes and therefore are also known as nuclear particles or fragments. A positive van den Bergh reaction occurs and the icterus index shows a slight increase.[5,6,20]

Multiple Myeloma

This condition is caused by a primary malignant tumor of the skeletal system which involves chiefly the active bone marrow. The skull, sternum, vertebra, pelvis and ribs are usually involved. Neurological and gastrointestinal disturbances are observed. Nausea, vomiting and colicky pain are the most common of the latter. There may occur a chronic nephritis. Examination of the spleen, liver, and lymph nodes shows metastases which also in rare cases may be present in the lungs. Bence-Jones protein is found in the urine. The marrow of the sternum contains plasmacytes in greater numbers than normal. Although most of these cases are accompanied by a normocytic anemia of the myelophthisic type a few do develop a macrocytic anemia. This disease is usually fatal. The only therapy which offers relief and promotes healing is deep x-ray.[8] Liver therapy does not appear to have any benefit.

Leukemias

Normocytic anemia of the myelophthisic type is usually associated with the aleukemic and subleukemic forms but a macrocytic anemia is sometimes observed. Examination of the marrow of the sternum reveals the presence of more megaloblasts and erythroblasts than is normal as well as a greater number of cells characteristic of the leukemia. It is believed that in both multiple myeloma and the leukemias the karyoblast division in the marrow is stimulated and the passage of the antipernicious anemia principle from the vessels is interrupted. Liver extract therapy has very little effect. It may provide some relief.

From the Editorial Research Department of the MEDICAL TIMES.

X-ray seems to be more effective than any other method.[8]

Normocytic Anemias

Accurate diagnosis of the normocytic anemias is extremely important and there are so many varied causes that no generalizations can be made as in the cases of the hyperchromic macrocytic and hypochromic microcytic anemias. In the latter anemias it is possible for a normocytic blood picture to be observed in mild cases or in cases where inadequate therapy has been instituted.

Many very serious and often fatal disease conditions are accompanied by this type of anemia in which neither the cell volume nor the diameter of the cell shows any serious change from normal. The color and saturation indexes are normal as well. There may be a distinct anisocytosis in some of this group of anemias. As the condition becomes more severe poikilocytosis, polychromatophilia, basophilic stippling or nucleated erythrocytes may be observed. There appears to be a direct inhibitory effect on the bone marrow function by the primary condition. Since the bone marrow is the site affected it follows that all of its elements would be involved as well. Consequently the condition is also accompanied by a leukopenia and thrombocytopenia (pancytopenia).[7]

In the therapy of these anemias it is first necessary to treat the primary disease and eliminate it if possible. If this is not possible then measures should be adopted which will provide as much relief to the patient as possible. Such therapy should serve as a protective to whatever remains of the normal bone marrow function of hemoglobin and red cell formation.

There are numerous causes for the normocytic anemias of which the major ones will be discussed here.

External and Internal Hemorrhages

Acute external hemorrhage occurs most frequently suddenly and may be caused by trauma, peptic ulcer, rupture of ectopic pregnancy, esophageal varices, typhoid ulcers, pulmonary tuberculosis, carcinoma of the gastrointestinal tract or the bronchi, aplastic anemia or the hemorrhagic diseases such as acute leukemia, hemophilia and purpura hemorrhagica.

The blood picture in such cases of acute posthemorrhagic anemia shows a high content of hemoglobin and a high red cell count as well as an increase in the erythrocyte volume and in the total number of leukocytes and an increase in the platelet count. Polymorphonuclear leukocytosis with a shift to the left is observed. At first the red cells are unchanged. The coagulation time is shortened and the blood volume decreased.[5,6]

In these cases the clinical symptoms manifested include low blood pressure with rapid, feeble pulse, restlessness, faintness or unconsciousness, cold clammy skin, rapid shallow breathing, which are more or less the symptoms of shock. Physiologically the body responds to this sudden loss of blood if the bone marrow is acting in a normal fashion by a contraction of the spleen as a result of which the red cells stored there are contributed to the circulating blood. Development of local areas of vasoconstriction takes place and the bone marrow immediately begins to increase its production of red blood cells. Several days are required before the latter production reaches its full capacity.

The first step in treating this condition is to stop the flow of blood and restore the blood volume by means of transfusion. A lack of blood to fill the vessels may cause death otherwise. When this has been done the blood picture shows a decreased hemoglobin content and red cell count and volume and a great increase in the total number of leukocytes and the platelet count. Reticulocytes are increased and there usually is macrocytosis and polychromatophilia with normoblasts. Hypercholesterolemia is frequently observed.

If the patient is normal as far as bone marrow capacity is concerned the blood picture should return to normal within 2 to 4 weeks. Regeneration of the blood may be slowed by the loss of iron which may occur in severe hemorrhages since the entire body only has available about 3 grams of iron. Therefore, in addition to liver extract therapy, iron is also needed.

Internal hemorrhage may be caused by either open or concealed bleeding. The anemia which results is known as chronic posthemorrhagic anemia and is caused by the bleeding over a period of time. Conditions such as pulmonary infarction,

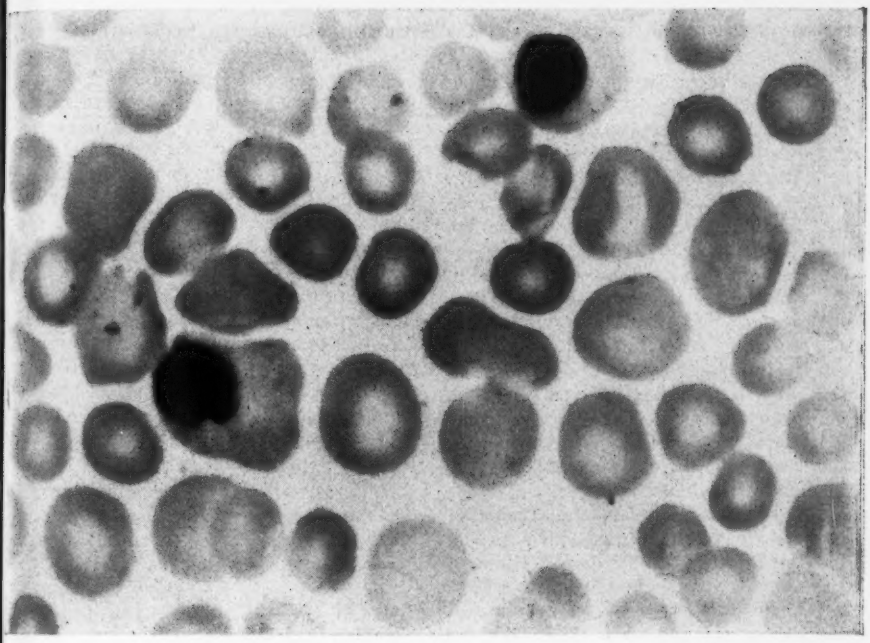

Fig. 1. Blood film of patient with pernicious anemia. Note variation in size and shape of erythrocytes, the predominance of macrocytes, a few red cells with nuclear fragments, and the two nucleated erythrocytes.

peptic ulcer, uterine bleeding, leaking aneuryism, recurrent and severe epistaxis or hemoptysis, malignant tumors, hemorrhoids and others may cause such internal hemorrhages. It is characterized by the same symptoms as is any severe anemia.

The blood picture shows a decrease of hemoglobin in each red cell. The red cell count is frequently reduced but may be normal or slightly increased. The color index is low. The platelet count is normal but the individual cells may be smaller than normal. Relative lymphocytosis frequently occurs. There may be some poikilocytes as well as erythrocytes and tiny microcytes in rings. The reticulocytes may be slightly less in number or normal. This is dependent upon the status of the bone marrow. Fragility of the red cell is normal. The plasma is pale and there may be an hypoproteinemia.

Therapy in such cases should first consist of elimination of the basic diseased

All illustrations courtesy of Sharp & Dohme.

venom and other hemolyzing poisons and parasites such as are present in malaria, condition since anemia therapy may hide the real cause of the condition. Transfusion is of value since the transportation of oxygen to the tissues is seriously affected by the loss of hemoglobin. The condition may also be complicated by infection, malnutrition and other conditions.[5,8] The treatment of the anemia is the same as for that resulting from external hemorrhages.

Internal Blood Destruction Anemias

The anemias which result from internal destruction of the blood include acute, subacute and chronic hemolytic anemias and acquired hemolytic jaundice. They are caused by incompatible blood transfusions, poisoning by metals such as lead, mercury, bismuth, gold or arsenic; organic poisons such as phosgene or benzene ring compounds or arsphenylhydrazine; severe bacterial infections in which toxins are liberated such as from the hemolyzing bacteria; extensive burns; intestinal parasites; snake

filaria, trypanosomiasis and relapsing fever.[5,6,8]

The blood picture in the chronic anemias shows a greatly reduced hemoglobin value of 3 to 5 Gm. In the acute types hemoglobinemia is present. The red cell count is reduced (below 1,000,000) as is the volume. The color index is low whereas the volume index may be normal or slightly lower. The red blood cells are usually normocytic but may be microcytic or macrocytic. Poikilocytosis is present accompanied by numerous normoblasts and macroblasts. There may be spherocytes. Stippling may be observed in the red cells and the reticulocytes are more numerous than usual. The red cell fragility is greater than normal. The total leukocyte content is increased to 30,00-50,000. Leukocytosis with myelocytes and sometimes myeloblasts are present. The platelets are frequently increased in size as is the count. Mild cases may have leukopenia. Examination of the bone marrow reveals a hyperplastic state. The icterus index is usually high. Bilirubin is sometimes produced in excess. Methemalbumin may be present in the plasma. Tests of the urine show albumin, absence of bile, urobilinogen and urobilin. The latter two may also be shown in the feces. Hemoglobinuria may take place.[5,21]

These anemias may be familial, which cases have greater chance of recovery because treatment is usually more adequate. The clinical symptoms include very dark feces and urine, semi-coma, extreme weakness and gastrointestinal symptoms dependent upon the speed of development.

The first step in the therapy is to remove the etiologic agent and then treat the patient for anemia. It may be necessary to give blood transfusions where the bone marrow is so seriously involved that it provides no red blood cells to the circulation. This sometimes causes an antihemolytic effect as well. Care must be taken in the transfusion for great damage can result if cold hemagglutinins, isohemolysins, and intragroup agglutinins are present. The extreme fragility of the red cells increases their susceptibility to hemolysis. It is suggested that the following precautions should be observed: employ high-titer typing serum; make both microscopic and macroscopic tests; test for autoagglutinins, and so forth; employ fresh citrated blood; be sure donor is of same blood group as patient, using group O only when the patient's blood is group O. It is important that transfusions be used sparingly so as to avoid any irreversible hemolytic trend resulting from the formation of isoagglutinins. The possibility of a serious reaction becomes greater with each transfusion in addition to the fact that a strain is placed on the heart, particularly in older patients. The symptoms of shock and a red cell count below 1,000,000 are indications for transfusion as well as preoperatively and postoperatively to splenectomy. Splenectomy is capable of causing recovery in six out of ten patients whereas the mortality rate otherwise is 100 per cent.[21]

In cases of chronic hemolytic anemia the splenectomy usually should be done between attacks.[20] In the meantime the anemia should be treated.

In severe cases liver extract should be given intramuscularly in the same dosage as in addisonian pernicious anemia. In milder cases oral therapy may be used.

Familial Hemolytic Icterus

Also known as congenital hemolytic jaundice this condition is included in the hereditary hemolytic anemias along with Cooley's and sickle-cell anemia. It is inherited as a true dominant character and usually appears during childhood or early adulthood.

The blood picture in this condition shows a moderate reduction in the erythrocyte count except in crises when it falls to 1,000,000. The erythrocyte volume is usually reduced; the hemoglobin content low and the color index below 1. The volume index may be normal or slightly below. The number of total leukocytes is usually normal as is the platelet count except during crises when it is raised. When there is a hemolytic crisis there is frequently an extensive leukocytosis. There is usually an increase in immature types as well as in lymphocytes, plasma cells and basophils. The leukocyte count returns to normal or slightly above normal when a remission occurs. The leukocytosis may be replaced by a leukopenia

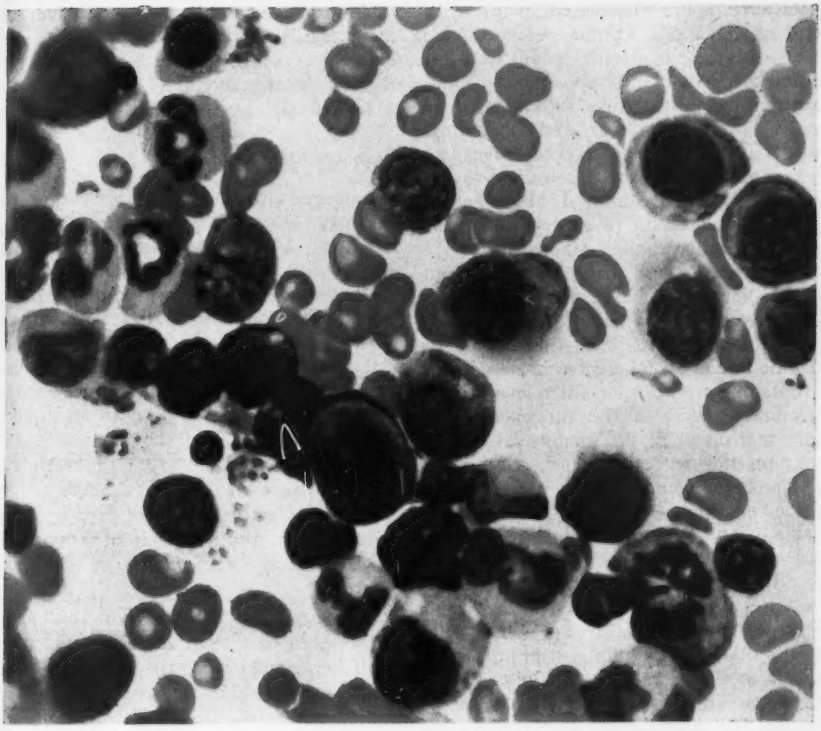

Fig. 2. Bone marrow film from patient with pernicious anemia showing numerous megaloblasts and mitotic figures, a few nucleated red cells and abnormal forms of the myeloid series. Marked variation in form and size of mature erythrocytes is observed also.

in some cases. The physical status of the erythrocytes is described as microspherocytosis. They are reduced in size, thicker than normal and spherical rather than biconcave. The number of microcytes and reticulocytes (sometimes 50 per cent) is increased. The fragility of the red blood cells is very great. A high icterus index and positive indirect van den Bergh reaction are noted.[5]

Clinically the following symptoms are noted: skin may be tinged yellow; weakness, lassitude, fatigue, upper abdominal pain and irregular menstrual periods in women. Fever, headache, fatigue and varying pains accompany each hemolytic crisis. There is no pruritus associated with the jaundice. The symptoms may first be noted upon removal to a high altitude, an increase in physical activity or following an acute illness.[5,6]

Every member of the family should be examined when one case in that family is ascertained. In those cases where it is mild and no anemia accompanies it no treatment is necessary unless the condition becomes worse. In severe cases splenectomy is the treatment of choice. Within two weeks the blood count is normal and the jaundice disappears although the blood smear remains abnormal and the fragility of the red blood cells persists. In some cases frequent intramuscular injections of liver extract have shown value. Iron, arsenic and vitamins have not elicited any definite response.

Sickle Cell Anemia

Sickle cell anemia, also known as African target-sickle-cell anemia, is also an hereditary condition usually found in the Negro race or those of mixed blood. It is clinically

characterized by rheumatoid pains with acute exacerbations, anemia, weakness, leg ulcers, shortness of breath, jaundice, pallor of mucous membranes, splenomegaly and recurrent hemolytic crises accompanied by nausea, vomiting, fever and abdominal pains. Those individuals who have had this condition are more sensitive to infections.[5,8,19] Many times it has been diagnosed as rheumatic fever, syphilis, tuberculosis or other similar conditions.

It has been found that 7 to 10 per cent of the Negro population possess this sickling phenomenon. There are several possibilities as to the cause of this disease. It may be due to an inherent weakness in the cytoplasmic network or there may be an hereditary defect in the function of the bone marrow or plasma changes may occur in connection with the oxygen tension.[5,8,19]

The hemoglobin content is reduced in sickle cell anemia. The red blood cells are sickle- and oat-shaped and show changes due to great blood destruction and rapid blood formation. The fragility is normal or slightly increased; reticulocytes are present. Observed also are target-cells, erythroblastosis, basophilic stippling, polychromatophilia and Howell-Jolly bodies. Between attacks the platelet count is normal but during crisis is increased. Leukocytosis with a shift to the left is observed as well as myelocytes, and an increase in monocytes. Eosinophilia may also be noted. Examination of the sternal bone marrow reveals an increase in the nucleated erythrocytes, particularly of normoblasts. The icterus index is increased and the van den Bergh reaction is positive. Urinalysis shows presence usually of albumin and casts and an increase in urobilinogen and urobilin accompanied by a fixed low specific gravity.

This condition runs a peculiar course. Repeated attacks and remissions frequently occur whereas in other cases the attacks gradually disappear. However, the sickle-cell still remains. In acute attacks, particularly in children, death may occur.[5,6,20,23]

Most therapeutic agents have proven of little value in this anemia. In severe crises transfusions have been of some value and in cases where these have occurred frequently splenectomy may help although it is not a cure for the basic cause. The diet should be adequate in protein and vitamins and iron therapy should supplement this.[20] Intramuscular doses of liver extracts as suggested for addisonian pernicious anemia should also be given for 3 to 5 weeks.

Splenomegalic Anemias

Included in this classification are the anemias associated with the following diseases: Hodgkin's disease; lymphosarcoma; Gaucher's disease; Banti's disease; cirrhosis of the liver; thrombosis of the portal or splenic veins; kala-azar; Neimann-Pick's disease; rickets; amyloidosis; tuberculosis of the spleen; and subacute bacterial endocarditis.[8] In this group enlargement of the spleen is the chief physical characteristic. The spleen is believed to be partially responsible for the resultant anemia due either to its excessive activity in destroying the red blood cells or by an alteration in the secretion of a hormone which affects the function of the bone marrow.[8]

Some of the members of this group have been discussed already and others will be omitted since they are not strictly anemic conditions but are disorders of other phases of the blood.

Those discussed here are also known as hepatic and lipoid anemias.[23] The anemia is usually moderate and the blood picture shows a red cell count of 2,000,000 to 5,000,000 and a somewhat below normal content of hemoglobin. The platelet count is usually low. Leukopenia of the neutropenic type is usually present. A normal reticulocyte count is usually observed accompanied by a few polychromatophilic cells and nucleated erythrocytes.[8]

Banti's disease or chronic congestive splenomegaly is caused by disturbances of the portal and venous circulation which are believed due to mechanical, toxic, or infectious factors. This condition may occur in childhood but is not really limited to any particular age. The mechanical factors which may cause this disease include thrombosis of the portal or splenic veins; cirrhosis of the liver; stenosis of the splenic vein; congenital narrowing of the portal bed; or an outside pressure causing ob-

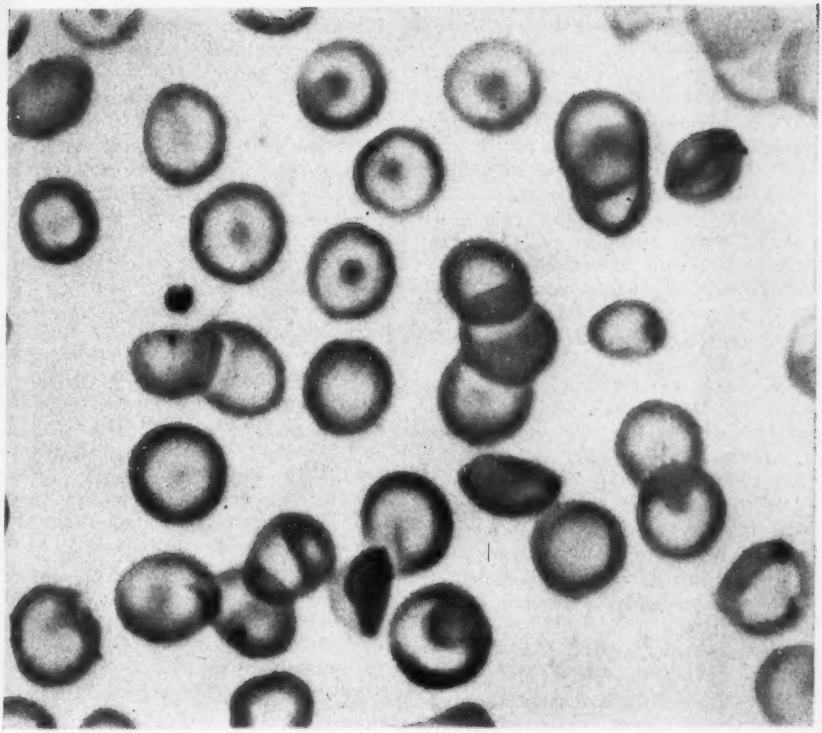

Fig. 3. Blood film of adult patient with mild form of Cooley's anemia or thalassanemia minor. Note the characteristic target cells, which are known also as Mexican hat cells and bull-eyed cells.

struction such as a gallstone in the common duct.

The blood picture in Banti's disease or chronic congestive splenomegaly shows an increase in hemoglobin in the individual cells or an hyperchromic anemia. The erythrocyte and leukocyte counts are decreased, whereas the color index and volume index are increased. The platelet count is usually reduced. A characteristic of this condition is a leukopenia with a decrease in neutrophils. Although classed with the splenomegalic diseases under the normocytic anemias the condition is usually macrocytic and hyperchromic. The spleen is enlarged, the liver is small and atrophy and hemorrhages sometimes take place.

The symptoms as manifested include abdominal pain or discomfort, hematemesis, chronic congestion of the spleen with fibrosis; weakness; general lassitude and portal hypertension.

The disease may be spontaneously arrested or it may be chronic but not disabling to the patient.[5,6,23,24] The first step in the treatment is to relieve the hepatic condition and stop its progress; treat the anemic condition and finally relieve certain symptoms such as the hematemesis. If massive anemia therapy is not sufficient then splenectomy is indicated. This is an aid in relieving the portal congestion by decreasing the blood flow in the portal vein and the discomfort caused by the enlarged spleen but there is a risk of fatality in 30 per cent of the cases as well as of peritonitis, liver insufficiency, peritoneal hemorrhage and mesenteric thrombosis.[24]

Niemann-Pick's disease is a constitutional, familial and congenital condition included in those diseases characterized by

splenomegaly. It is usually found in infants and children and particularly in those of Jewish origin. More women than men show symptoms of this condition. This disease is uniformly fatal and is characterized by a lipoid histiocytosis. It resembles Gaucher's disease but is earlier in onset and more rapid in its course.

The blood picture in this condition shows somewhat reduced red cell and platelet counts with leukopenia and relative lymphocytosis and monocytosis. Occasionally leukocytosis is observed. The anemia is moderate. The blood cholesterol may be increased. Primarily this condition is a disease of lipoid metabolism.

Clinically the symptoms include diffuse brownish discoloration of the skin; slowing of mental and physical growth; frequently a mongoloid appearance; development of minor infections; involvement of liver and spleen; inability to digest food; general emaciation; leukocytosis and an unaccountable fever. Splenectomy has not been found to either cure or arrest the progress of the disease.[5,6,24,25]

Gaucher's disease is characterized by an excessively enlarged spleen and liver. Exposed portions of the body show a brownish pigmentation. There are blood changes and osteoplastic changes in the bones. The conjunctiva near the cornea of the eyes shows a wedge-shaped thickening. This condition also is prevalent in children and young adults, particularly females and people of Jewish origin. It is familial but not hereditary and is rarely fatal except in very young children. It may last for many years with remissions. The cause of this disease is believed to be a disturbance of lipoid (kerasin and sphingosine) metabolism. The lipoid is phagocytized by monocytic cells resulting in foam cells which are found when a sternal or splenic puncture is made. The foam cells are an aid to the proper diagnosis.[5,6,8,24]

The blood picture in Gaucher's disease shows decreased red cell and platelet counts. Leukopenia with a relative lymphocytosis commonly takes place. Monocytosis may occur. It is a moderate normocytic anemia with very little indication of polychromatophilia or erythroblastosis which would show evidence of active blood regeneration. The spleen contains large cells consisting of kerasin.

Irradiation of the spleen and the bone lesions has not been effective in altering the course of the disease. It does reduce the size of the spleen.[24] Splenectomy is of some value in ridding the patient of a bulky organ and in improving the blood picture but it does not alter the course of the disease. In some cases the bones have become worse. The mortality rate is 20 per cent.[24,25]

Myelophthisic Anemias

The following diseases have associated with them a myelophthisic anemia: leukemias; metastasis of malignant tumors to bone; multiple myeloma; osteomyelitis; osteosclerosis (Albers-Schonberg disease); xanthomatosis (Hand-Schüller-Christian's disease).[8]

Invasion and destruction by other tissues of the bone marrow is responsible for these anemias. The marrow is therefore deficient but that portion which remains is stimulated by the anemia to a rapid production of red blood cells. Thus there is a deficiency of non-nucleated erythrocytes associated with an increased number of reticulocytes.

In the leukemias the anemia is partially caused by phagocytosis of red blood cells by the leukemic cells.

Multiple myeloma is usually characterized by a normocytic anemia although occasionally by a macrocytic anemia under which it was discussed.

Malignant tumors are usually not the cause of anemia unless they become infected, bleed or invade the marrow.[8]

Hand-Schüller-Christian's disease is a disorder of the fat metabolism which is characterized by a retention of cholesterol. Deposits of cholesterol esters are frequently found in the organs containing the reticulo-endothelial cells. It is usually found in children and is evidenced by diabetes insipidus; xanthomatosis; cranial defects visible in roentgenograms and detected by palpation; lipoid granulomatous masses which particularly involve the bones, brain, liver and spleen; sometimes an enlarged liver and spleen; exophthalmos as a result of invasion of the orbits.

The blood picture in this condition shows a reduced platelet count and an occasional leukopenia. The anemia is not

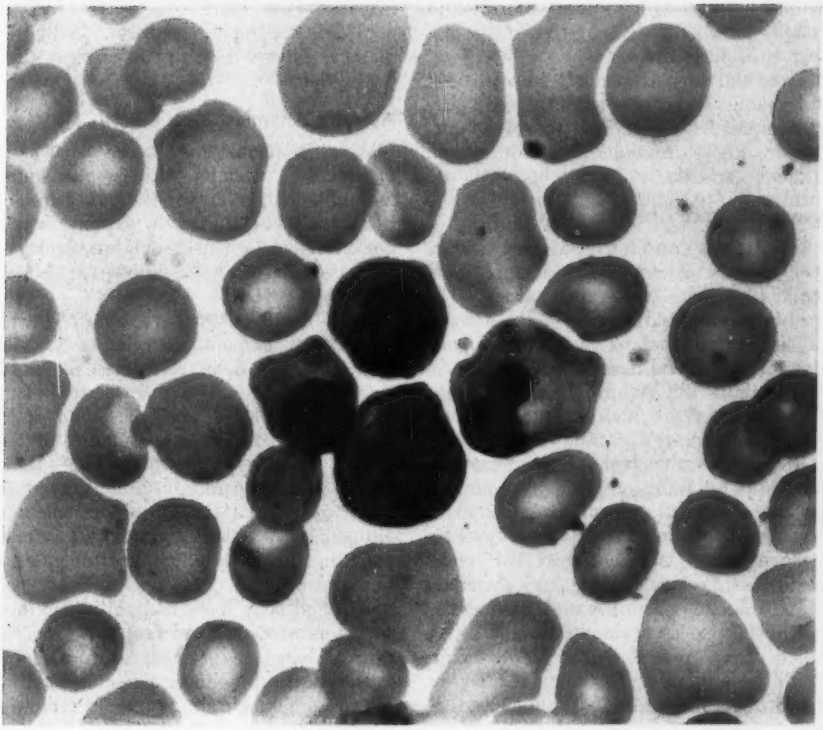

Fig. 4. Blood film of infant with erythroblastosis or hemolytic disease of the newborn. The cells in the central group of normoblasts are in various stages of development. The mature erythrocytes are macrocytic and in general are well filled with hemoglobin.

always severe and is not always present. It may be an aplastic type. There is usually a decrease in blood cholesterol, fatty acids and phospholipids.

Roentgen ray therapy has been found to be of some value in improving those cases in which there is an eosinophilic granuloma of the bone. No other therapy is effective.

Anemias Caused by Deficiency Diseases

Normocytic anemia is frequently associated with the following deficiency diseases: cretinism; myxedema; pituitary cachexia; scurvy; Addison's disease; pellagra and sprue.[8] Sprue and pellagra have been discussed with the macrocytic anemias although they are more frequently accompanied by normocytic anemias.

The anemia associated with cretinism and myxedema is caused by a deficiency of thyroid, particularly after this has been extended over a period of time. It is corrected by the administration of thyroid or thyroxin. In pituitary cachexia (Simmonds' disease) anterior pituitary hormones are given since this is evidence of a deficiency of these hormones. Thyroid or thyroxin is also of value as iron may also be.

Vitamin C or ascorbic acid is specific in the anemia of scurvy which is due to a deficiency in the red blood cell formation and to internal hemorrhage. Urobilinogen and the icterus index are frequently above normal.

Very few patients with Addison's disease develop anemia and in those cases which do, it is usually mild. Administration of adrenal cortex or desoxycorticosterone acetate and maintenance of the

sodium balance with sodium chloride and sodium bicarbonate is effective. Liver extract is of little use.

Liver and stomach concentrate with iron and vitamin B complex administered orally will improve more rapidly the blood picture in scurvy, myxedema, cretinism and pituitary cachexia.

Anemias Caused by Infection

Infection is considered to cause normocytic anemia or any other anemia more commonly than any other agent. The anemia is produced in one of three ways; (a) inhibition of the active bone marrow; (b) internal blood destruction; or (c) interference in the maturing process of the normoblast to the non-nucleated erythrocyte. The severity, location and duration of the infection control the degree of the anemia. The infections which commonly cause a normocytic anemia are: hemolytic organisms; glomerular nephritis; pyelitis; pyelonephritis; subacute bacterial endocarditis; sinusitis; cholecystitis; rheumatic fever; infectious arthritis and bacteremia.[8]

Known also as a simple chronic or so-called secondary anemia the blood picture in this condition shows a moderately reduced red cell count of 2,000,000 to 4,500,000 with a reduced erythrocyte volume and reduced hemoglobin (6 to 12 Gm.) value. The total number of leukocytes is usually increased to some extent and the platelet count may be reduced. Leukocytosis to a slight extent appears accompanied by an absolute increase of lymphocytes. Severe infections may cause such an increase in white cells as to appear leukemic. Commonly observed in the red cells is anisocytosis with poikilocytosis to a slight extent. Cases of severe infection may be followed by granulopenia, thrombopenia and erythropenia as a result of inhibition of the bone marrow. This may replace the leukocytosis. In severe thrombopenia or hemolysis, hemorrhage may take place. The blood plasma is normal in color. The nature of the anemia may be microcytic instead of the usual normocytic type.

This anemia may also be due to infestations or other systemic diseases. Clinical evidence of its presence is lassitude, pallor and the common symptoms of anemia.[5,6,24]

Therapy in these cases consists of removal of the focus of infection accompanied by supportive treatment, chiefly iron therapy. This will be discussed more thoroughly later.

Aplastic Anemias

Aplastic anemia may occur as a result of intensive therapy with arsphenamine, radium, roentgen-ray or the sulfonamides. It may also be caused by chronic benzol poisoning, malignant neoplasms, leukemia and osteosclerosis, also known as Albers-Schönberg disease.

The blood picture in aplastic anemia shows a low hemoglobin value of 3-6 Gm. per 100 cc. and an erythrocyte count of about 2,000,000. The erythrocyte volume is usually reduced and the color index usually normal. The total number of leukocytes is reduced to 1500. The platelet count is also reduced. Granulocytes are either greatly reduced in number or absent altogether. A relative lymphocytosis takes place and an absolute lymphocytopenia may be present.

The red cells show basophilic stippling, polychromatophilia and few or no reticulocytes. There may also occur a moderate anisocytosis, poikilocytosis and erythroblastosis. The bleeding time is somewhat prolonged and the clot shows poor retraction. The blood pictures of aplastic anemia and aleukemic leukemias are so similar that a differential diagnosis can only be done by examining the sternal marrow. Practically no nucleated cells are observed in the former whereas they are numerous in the latter.

In aplastic anemia the sternal bone marrow is composed chiefly of red corpuscles. The nucleated cells present are chiefly lymphocytes. The marrow may be in a condition known as marrow block in which it is histologically normal but its function is defective. It may be fatty and drained of cells.[5,6]

Aplastic anemia may be idiopathic or secondary. The congenital type is relatively rare but if present is evidenced at about 3 months of age. The symptoms of aplastic anemia include weakness; fatigue; pallor which increases; possibly purpuric spots; oozing from gums and soft tissues of mouth; and excessive uterine bleeding. If the anemia is severe palpitation and hemic murmur may occur.

Both the congenital and acquired types of aplastic anemia have a grave prognosis. Death usually follows diagnosis of the congenital type in a few weeks. Intensive therapy has brought about recovery in some acquired type cases.

The first step in therapy is to remove the causative agent of the condition and then institute persistent treatment. Some workers believe that there is always a hope for an aplastic or hypoplastic marrow to again assume its normal function. Blood transfusions at frequent intervals are the main treatment. Liver extract therapy and pentnucleotide bone-marrow therapy should be employed as supportive measures. Marrow transfusion should be investigated further. X-ray in stimulating doses is useful only in cases where the marrow is not aplastic. Transfusions, liver extract, iron and vitamin therapy over periods of months or years have brought about recovery in chronic hypoplastic anemia.[20, 26]

Hypochromic Microcytic Anemias

The hypochromic microcytic anemias are also known as the iron deficiency anemias and usually occur as a result of insufficient iron so that there is a shortage of hemoglobin. This results in a deficient development of the red blood cells in the bone marrow. In many cases there may be no change in the size of the red blood cells. The presence of microcytes in this type of anemia is much less frequent than the appearance of macrocytes in the hyperchromic anemias.

The most common characteristics of the blood picture are a low hemoglobin content with a low red blood cell count. The remainder of the picture has been described under the normocytic anemias in the anemia caused by infections also known as simple chronic or secondary anemia. It is a question as to which classification should possess this particular anemia since it falls under both.

The hypochromic microcytic anemias include the anemia which occurs as a result of chronic hemorrhage due to carcinoma; peptic ulcer; hemorrhoids; hookworm infestation; ulcerative colitis and amebiasis as well as hemorrhages from the urinary and respiratory tract; idiopathic hypochromic anemia; and the hypochromic anemia from dietary deficiencies. Included in this latter group are anemia of milk-fed children; microcytic anemia of pregnancy; chlorosis; starvation and adult nutritional anemia.[8]

As previously stated the soluble ferrous salts are the most easily absorbed and assimilated in the upper part of the small intestine. It is thought that other forms of iron first must be reduced to the ferrous state before they can be utilized. A comparison of the various iron salts has shown that a dosage of 0.8 Gm. (12 gr.) of ferrous salt is the equivalent of 3.0 Gm. (45 gr.) of reduced iron; 4.0 Gm. (60 gr.) of ferrous carbonate in Blaud's pill; and 6.0 Gm. (90 gr.) of ferric ammonium citrate. For children the average dosage of 0.6 to 0.8 Gm. (10 to 12 gr.) of ferrous sulfate is the equivalent of 4 to 6 Gm. (60 to 90 gr.) of ferric ammonium citrate. In infants up to 4 years of age the average dosage of 0.4 to 0.5 Gm. (6 to 8 gr.) of ferrous sulfate is equivalent to 1 to 3 Gm. (15 to 45 gr.) of ferric ammonium citrate and 0.15 Gm. (2½ gr.) of ferric pyrophosphate.[26a]

Anemia due to Chronic Hemorrhage

Hypochromic microcytic anemia is most commonly caused by chronic hemorrhage. The intake of iron each day is very little in excess of the body's requirement. Therefore an excessive loss of blood removes considerable iron from the reserve supply and consequently an iron deficiency results. Since iron is so necessary to hemoglobin formation it can be clearly seen how this starts the chain of circumstances resulting in anemia. In normal cases of moderate bleeding the blood-forming tissues automatically respond but in repeated bleeding the depletion of iron hinders this process.

The first step in treatment, of course, is to stop the hemorrhage. Iron therapy should then be instituted. It may be given in the form of ferrous sulfate. Response to this is so rapid that it is considered specific. The diet should be rich in iron. Administration of vitamin B complex may prove helpful. Parenteral iron is not only costly but not necessary. There is considerable controversy over the question of administration of liver extract in the iron deficiency ane-

mias. Some[4, 7, 8, 15] recommend its use whereas others state that it has little or no effect.

Idiopathic Hypochromic Anemia

Idiopathic hypochromic anemia most frequently occurs in middle-aged women but may also be observed in men as well. Macrocytosis is often observed. The symptoms are typical of anemia but also include atropic glossitis, achlorhydria and spooning and recurving of the fingernails. In the true sense of the word the condition is not idiopathic. However, it has been termed this since two or more of the following factors are always present: (a) a poor appetite for meats results in an insufficiency of iron; (b) achlorhydria and poor gastric digestion hinder the absorption of iron due to the inability to liberate inorganic iron from its organic compounds; (c) chronic hemorrhage, usually menorrhagia.

The decrease in average cell volume and the minus one color index differentiates this disease from addisonian pernicious anemia. The antipernicious anemia principle is believed to have value although it is not specific.

Ferrous sulfate in doses of 20 or more grains should be given three times daily. When the blood picture returns to normal the dosage is reduced to 10 grains three times daily. Continuous treatment for the remainder of the patient's life is necessary. All causative factors such as hemorrhage, gastritis, septic foci, or achlorhydria should be removed. Pregnancy should be avoided. The diet should be corrected. Glutamic acid hydrochloride may be given to relieve the achlorhydria. Local treatment of esophageal dysfunction or abnormalities may be necessary when such conditions are present.

Dietary Deficiency Anemia

Dietary deficiency anemia occurs in cases where patients are on a milk diet for any length of time. This is apt to occur in infants who are continued on a milk diet for a longer period of time than normal or in premature infants where no excess reserve of iron was stored in the blood before birth; and in peptic ulcer patients on a milk and cream diet without supplemental iron.

Children may develop anemia from many causes. This should of course be treated by removal of the etiologic disease and supportive treatment as in adults.

Nutritional anemia in children is the same as in adults. If the dietary deficiency either of the mother during pregnancy or of the infant after birth has brought about a deficiency disease such as rickets, this must be corrected by the use of specific vitamin therapy. Tetany in newborn infants may be related to hypoparathyroidism but after the neonatal period vitamin D deficiency is the etiologic factor. Tetany may be accompanied by rickets. Prophylaxis against this should be begun at the age of 2 weeks with a daily dose of 10 drops of a vitamin D concentrate for one year. In therapy the dose is 60 drops daily for 1 month reduced to 10 drops after that period. Scurvy often occurs in infants given boiled milk without vitamin C supplements. Vitamin C in orange juice or as the synthetic should be given daily in doses up to 50 mg. beginning the second month of life as prophylaxis. Therapeutic daily doses of 100 mg. of ascorbic acid should be given for 2 weeks and then reduced to a maintenance dose in cases of scurvy. In many cases the anemia will also improve. However, more rapid recovery will result from antianemia therapy.

A daily dosage of 1 mg. of elemental copper to 25 mg. of elemental iron in milk for infants and small children is recommended.[27] Larger doses are necessary for older children and adults.

Microcytic Anemia of Pregnancy

This anemia is differentiated from the macrocytic anemia of pregnancy by the low color index. It occurs in the latter months of gestation and is caused by the requirement of the fetus for extra iron to be stored in the liver. Hypo- or achlorhydria and a deficient intake of the extrinsic factor may also cause it. Therapy of this condition is the same as for the others. Larger doses may be required due to the need of two individuals.

Chlorosis

Chlorosis is also an iron deficiency anemia. The hemoglobin content of the red blood cells is considerably reduced. The erythrocyte count is reduced depen-

dent upon the severity of the disease. The erythrocyte volume is reduced and the color index is low. The red blood cells are usually microcytic. Polychromatophilia and poikilocytosis may occur. The leukocyte count is usually normal, varying only when there is an accompanying infection. The platelet count is usually normal but may be reduced and the individual elements may be smaller in size. The blood plasma is very pale. The sternal bone marrow becomes hyperplastic and the small polychromatophilic normoblasts increase in number. Achlorhydria or hypochlorhydria takes place.

Chlorosis very often occurs as a result of achlorhydria in the gastric juice, thus preventing digestion of foods containing iron.

It is characterized by weakness, dyspnea, irregularity of menstruation, green pallor and vasomotor disturbances. Prompt response to oral inorganic iron therapy is noted. This condition is not so common as it was years ago. It very rarely occurs in men.[5, 6, 20]

Starvation Anemia

Anemia rarely follows starvation due to the fact that the body conserves its reserves of iron when the supply is eliminated. However, other factors may interfere with the absorption of iron from the intestinal tract. For example, large quantities of fixed alkali in the stomach may interfere. The cause should first be eliminated and then antianemia therapy instituted.[8]

In all hypochromic microcytic anemias it is important that the iron given be in the inorganic form such as ferrous sulfate, ferrous carbonate or the complex ferric ammonium citrate. The daily dosage should be 3 to 6 grams.

In every case of anemia it is important to remember that the type should be ascertained and then the cause. When these two factors are known rational therapy can be instituted.

Anemias in Infancy and Childhood

The anemias to which children are most susceptible have been discussed under the respective classifications. In children there are physiologic and anatomic changes taking place in the body and within the hematopoietic system. Consequently in diagnosing anemias in young patients these normal changes in the system must be considered.

Classification

A simplified classification has been made of the anemia occurring in infants and children.[28] It is based on the etiologic factors and is as follows:

1. Anemia secondary to infection
2. Dietary anemia (nutritional or alimentary anemia)
   Iron deficiency during:
   - (a) Prenatal period
   - (b) Postnatal period
3. Physiologic anemia of prematurity
4. Congenital and familial anemias:
   - (a) Congenital hemolytic jaundice
   - (b) Sickle cell anemia
   - (c) Mediterranean anemia
5. Erythroblastosis (acquired hemolytic states)
6. Primary diseases of the blood:
   - (a) Aplastic anemia
   - (b) Purpura
   - (c) Hemophilia
   - (d) Leukemia
   - (e) Lymphoblastoma (Hodgkin's disease, lymphoma, etc.)
7. Anemia secondary to hemorrhage
8. Miscellaneous
   - (a) Anemia associated with chemical poison, such as benzene, lead, acetanilid and sulfonamide compounds
   - (b) Anemia associated with parasitic infestation
   - (c) Anemia associated with neoplasms

From a study of 1500 children it was found that 34 per cent were anemic and that the second year of life is the time of greatest incidence. The causative factor in the 514 patients with anemia included infection, deficient diet, prematurity or combinations of two or of the three factors.[28]

Any one of three types of anemia may appear as secondary to an infection. There may be a decrease in the production of red blood cells, thus showing an hypoplastic or aplastic anemia. Secondly the circulating red blood cells may be destroyed at a greater rate, thus causing an hemolytic anemia. The third type is the secondary

thrombopenic purpura in which there is a decreased production or increased destruction of blood platelets.

Iron deficiency is usually the basic factor responsible for dietary anemia. This may be due to a diet containing insufficient sources of iron or to an inability to absorb or utilize the iron during the prenatal or postnatal periods. This type is considered as an hypochromic microcytic anemia. In those cases where it is due to an insufficient quantity of the antipernicious anemia or erythrocyte-maturing factor in the liver a macrocytic anemia develops. Folic acid is employed in treating this condition.

All infants undergo an anemia during the neonatal period and if born prematurely show evidence of this anemia. If after birth the infant contracts an infection or is given a deficient diet a more severe anemia will develop.

Included in the congenital anemias are those in which abnormal red cells are formed as a result of some disturbance of the forming organs. These anemias may also be familial and racial.

The acquired hemolytic states, of which erythroblastosis is the most common, include other more rare conditions. The causes listed under 6, 7 and 8 are rare in infants and children but must be considered in cases difficult of diagnosis.[28]

Normal Blood Picture

The normal blood picture in infants and children shows a red cell count of 5,500,000 per cu. mm. the first day of life. In the second week this is lowered to 5,000,000 and later in life to 4,000,000 per cu. mm. as the lower limit of normal. Hemoglobin content the first day is 20 Gm. which is lowered in 2 weeks to 17 Gm.; in first 2 years, 10 to 12.5 Gm.; 3 to 5 years, 12.5 to 13 Gm.; 5 to 10 years, 13 to 13.5 Gm.; and 10 years to puberty, 13.5 to 14.5 Gm. At birth the nucleated red cells range from 3 to 10 per 100 white cells. From 0.5 to 1.5 per cent of reticulocytes are present. This value falls below 0.5 per cent in hypoplastic and aplastic anemia and increases in hemolytic anemia. Therapy of the deficiency anemia raises the level. The volume of packed red cells in infants and children is at least 36 per cent and in older age groups is at least 40 per cent. The normal icterus index is 4 to 6 units whereas in hemolytic anemias it is 15 to 25 per cent. The fragility test shows a normal range of 0.425 to 0.325 per cent sodium chloride. In sickle cell, Mediterranean and iron deficiency anemias this is decreased whereas in congenital hemolytic jaundice and in some cases of acute hemolytic anemia it is increased.[29]

Blood Picture in Anemias

Certain diagnostic features of the anemias are seen in a study of the blood smear. In erythroblastosis there are nucleated red blood cells and hyperchromic macrocytes. Hemorrhagic anemias, in which are included the hemorrhagic disease of the newborn, hemophilia, thrombocytopenic purpura, more rare hemorrhagic disorders and nonspecific diseases associated with blood loss, show normochromic and normocytic red cells in acute blood loss. In chronic blood loss the red cells are hypochromic microcytes.

The iron deficiency anemia is characterized by hypochromic microcytes, poikilocytes and pronounced paleness in center of red blood cells. The anemia of prematurity in the early stages shows normocytic and normochromic red cells which later develop into hypochromic microcytes. Extremely large macrocytes, microcytes, abnormal neutrophils and occasional nucleated red blood cells are observed in the megaloblastic anemia of infancy. Normochromic and normocytic red blood cells characterize hypoplastic and aplastic anemia. In congenital hemolytic anemia there are present spherocytes and a large number of reticulocytes. Sickle cells, target and oval cells and hypochromic macrocytes are characteristic of sickle cell anemia. The severe type of Mediterranean or Cooley's anemia shows extremely large, thin hypochromic macrocytes, nucleated red blood cells and pronounced poikilocytosis and anisocytosis. The blood picture in the mild type shows hypochromic macrocytes, basophilic stippling, polycythemia and target and oval cells. The red blood cells have an increased resistance to hemolysis when placed in hypotonic solutions of sodium chloride in both types.[29]

Bone Marrow in Anemias

The hemolytic group of anemias includes erythroblastosis fatalis, congenital hemolytic jaundice, sickle cell anemia, Mediterranean anemia and acute hemolytic anemia. The bone marrow in these conditions is hyperplastic with an increase of nucleated red cells, particularly the normoblasts. Granulopoiesis may also be active. The marrow is hyperplastic with an increase in normoblasts but granulopoiesis is unchanged in the iron deficiency anemias.

Hemorrhagic anemia is characterized by an hyperplastic marrow with an increase in the nucleated red cells, particularly the normoblasts. In chronic blood loss granulopoiesis is unchanged whereas in acute hemorrhage it is active.

The blood marrow pattern of megaloblastic anemia of infancy resembles that of pernicious anemia in relapse. The erythropoiesis is megaloblastic and certain changes take place in the granulocytes as in pernicious anemia. The myeloid elements, megakaryocytes and normoblasts are sharply reduced and the lymphocytes increased in aplastic anemia. Frequently there is a decreased cellularity. An hypoplastic marrow with a definite depression affecting chiefly the normoblasts is characteristic of hypoplastic anemia. Eosinophils and primitive cells such as hemotogones are increased. In lmphatic leukemia lymphoblasts and lymphocytes replace the normal nucleated elements. Moderate hyperplasia of the bone marrow and an increase in the megakaryocytes are noted in thrombocytopenic pupura.[29]

Origin of Anemia

Two basic mechanisms which are responsible for the anemia of infancy and childhood are diminished production of hemoglobin by the marrow and excessive loss of hemoglobin from the circulation. These of course are also the basic mechanism causing anemia in adults and their principles are similar. In infants and children, however, certain peculiarities are present as a result of the effects of growth, metabolism, nutrition and susceptibility to infection. Not only do these factors modify the anemia but also are favorable to its development.[30]

Therapy of Anemias in Infancy and Childhood

The treatment of erythroblastosis fetalis has already been discussed. Slipped cord ligature, trauma or asphyxial bleeding are usually responsible for hemorrhage in the newborn. Elimination of asphyxial states reduces the incidence. In the former transfusion is valuable. Hypoprothrombinemia neonatorum practically has been eliminated by administration of vitamin K to the mother and child. Transfusion of blood is indicated in hemophilia and in congenital thrombocytopenic purpura. If the latter cannot be controlled in this manner splenectomy is necessary.

Chemotherapy and transfusion of blood are indicated in infections accompanying bacteremia or acute hemolytic anemia or in bleeding and purpura caused by a depression of platelets. This condition is no longer very common. No known cause has been found for chronic hypoplastic anemia which takes place in the latter part of the neonatal period. Blood transfusion appears to be the only affective treatment. Splenectomy is still without sufficient evidence of its value. Some advocate the use of marrow stimulants.[31]

The anemias which occur in children of one month to two years are usually caused by iron deficiency. It is possible that other elements needed for the synthesis of hemoglobin such as chlorophyll, copper, trace elements and protein may also be lacking. However, this does not necessarily indicate the use of the many preparations containing numerous materials.

Copper is known to be necessary as a catalyst in hemoglobin synthesis and to be involved in iron utilization. However, supplementary copper therapy is not necessary as a routine but only in those cases where the infant was given a milk diet exclusively or where the condition does not respond to iron therapy and there are no other conditions affecting this. The usual dosage of copper in such cases is 1 cc. per kilogram of body weight of a 0.5 per cent solution of copper sulfate. Older children receive smaller doses.

Routine use of liver fraction is not necessary since the status of liver in hypochromic microcytic anemias in children has

not been established. Vitamins are only necessary for good nutritional balance. Pureed liver, in doses of 20 Gm. daily up to 2 years of age and 40 Gm. to children of 2 to 6 years, may be given.[31]

In iron deficiency it is necessary to ascertain all of the causes and eliminate them as well. Such causes may include failure of absorption and utilization of necessary materials or the iron reserve may be deficient due to maternal anemia during gestation, premature and multiple births or premature ligation of the umbilical cord. Since there is a need for more iron to compensate for the increased blood volume the onset is hastened and rapidly becomes more severe.

A typical prescription for the administration of ferrous iron is as follows:

Ferrous sulfate	9.0 Gm.
Spirit of peppermint	1.0 cc.
Solution of amaranth	1.0 cc.
Syrup of citric acid	60.0 cc.
Simple syrup to make	120.0 cc.

Four cc. of this preparation provides approximately 0.16 Gm. (2.5 gr.) of anhydrous ferrous sulfate. Acid vehicles are used since acid facilitates ionization of the iron. Absorption of iron may be supplemented by use of reducing agents such as ascorbic acid or citrus fruits. The ferrous salts are preferable since smaller doses can be given and thus there is less irritation in the gastrointestinal tract. Ferrous gluconate is preferred by some.[31]

Although the dosage of iron preparations is subject to wide variations the following schedule of doses for ferrous sulfate is recommended as being effective: birth to 2 years, 0.26-0.39 Gm. (4-6 gr.) daily; 2 to 6 years, 0.39 Gm.-0.52 Gm. (6-8 gr.) daily; and 6 to 12 years, 0.65 Gm.-0.78 Gm. (10-12 grs.) daily. If ferric and ammonium citrate is preferred it should be administered in the form of a 10 per cent solution in daily doses of 2 cc. per Kg. of body weight. Small doses should be given at first and gradually increased. A reticulocyte count will show how effective the therapy is.[31]

The reticulocytes should begin to rise within three days of institution of therapy and a peak is reached in seven to ten days. This peak is usually in an inverse ratio to the level of hemoglobin. The lower the beginning hemoglobin value the higher will be the reticulocyte response. As normal hemoglobin values are approached the rate of increase in hemoglobin slows considerably.

The dosages presented here and elsewhere in this article for ferrous sulfate may seem excessive but it has been shown that large doses are necessary and that the body utilizes only the amount it requires and excretes the excess.

Pernicious anemia is relatively rare in children under 12 although states resembling this condition have been observed. A megaloblastic anemia accompanied by leukopenia with degenerative changes in the granulocytes and thrombopenia has been reported. This has resulted from infections of the upper respiratory tract or a nutritional disturbance of some type. A response of the reticulocytes was noted when folic acid or liver extract was administered. Relapses do not occur following adequate therapy. Folic acid is administered orally in daily doses of 5 to 20 mg. and intramuscularly in doses of 1 to 2 mg. Crude liver extract is given in doses of ½ to 2 units daily.[31]

Liver, folic acid or iron have no effect in the anemia accompanying scurvy unless there are other complications. Specific for this condition are ascorbic acid or citrus fruit juices. An immediate reticulocytosis occurs upon institution of such therapy.

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CONTEMPORARY PROGRESS

NEUROLOGY

The Effect of Oral Administration of Lactic Acid Upon the Clinical Course of Depressive States

H. Lowenbach and M. H. Greenhill (*Journal of Nervous and Mental Diseases* 105:343, April 1947) report the use of lactic acid in the treatment of depressive states. The mode of action of convulsive therapy on mental disorders has not yet been determined. The lactic acid therapy was employed on the theory that the increase of lactic acid in the blood caused by the violent muscular activity during an induced convulsion may be a factor in the therapeutic effect of the convulsion. The mixture employed in treatment contained 25 cc. of lactic acid (U.S.P.) and 25 cc. of sodium lactate (60 per cent) in 300 cc. milk. The patients given this treatment were told that it was of an experimental nature, that it had benefited some patients but not all, that it was in no way harmful, and that their cooperation was necessary. Lactic acid therapy has been used for more than 100 patients, but only 45 of these have been studied under controlled conditions and adequately followed up for at least fifteen months. All the patients treated showed a definite depressive state, but no distinction was made between the types of depression in this series. Patients showing a marked sleep disturbance were given the lactic acid mixture just before retiring; sedation was discontinued. If tension symptoms predominated the mixture was given early in the morning and breakfast deferred two hours. Most patients suffer temporary gastric discomfort after taking the lactic acid mixture, but only a few were nauseated. The treatment had a definitely favorable effect on the sleep pattern of those who had sleep disturbances. Of the 45 patients treated with lactic acid, 7 have recovered from the depressive state, 5 show marked improvement, and 14 a lesser degree of improvement. All these 26 patients were able to return home and resume their activities; 3 of these patients had a recurrence of depression within a year, and returned to the hospital for electric shock therapy; the others have continued to do well, some for three years. Of the remaining 19 patients in the series, 8 showed temporary improvement under lactic acid therapy but required electric shock therapy; 7 showed no improvement on lactic acid therapy but recovered with electric shock therapy; 4 failed to recover with either lactic acid or electric shock therapy. These results indicate that since the oral administration of lactic acid and sodium lactate exerts some influence on the depressive states, the role of carbohydrate metabolism in these states deserves further investigation. It may be that lactic acid is "the common denominator" in convulsive therapy and in other methods of physiotherapy that have been found of value in depressive states.

COMMENT

Such experimental attempts deserve recognition, whether further clinical trial shows it to be less auspicious than as now presented or not. Certainly the attack on the problem of mental illness has changed rapidly to an aggressive phase from that of the passive, philosophical, and mere custodial attention to the patient.

Any method whereby the number of patients admitted to institutions can be reduced, and the patients treated in their own environment, deserves encouragement; deserves the publicity devoted to neuropsychiatric disorders in the second World War, for a great deal remains to be done to break down the laity's feeling of "stigma" concerning mental disease. Hence welcome will be extended to a treatment which permits patients to be treated in their home environment, as are sufferers from heart or kidney disease.

—H.R.M.

Use of d-Tubocurarine Chloride and Thiopentone in Electro-Convulsion Therapy

J. A. Robson and Frederick Prescott (*British Medical Journal* 1:445, April 5, 1947) report the use of *d*-tubocurarine chloride and thiopentone (pentothal) in electric shock therapy. Thiopentone was used for preliminary anesthetization, the usual dose being 0.3 gm; the usual dose of *d*-tubocurarine required to give adequate relaxation was 0.3 mg. per kg. body weight, but the dose was carefully individualized in each case so as to give adequate relaxation but not depress respiration too deeply. The electric shock was given three to five minutes after the injection of *d*-tubocurarine. This method has been employed in 50 patients, for most of whom unmodified electro-convulsion therapy would have been dangerous. In the curarized patient under electric shock therapy, the muscles of the eyelids and face twitch, but there is little movement of the limbs and trunk. Immediately after administering the electric shock, the patient gives a slight jerk, corresponding to the tonic convulsion, and the clonic convulsion is greatly modified without gross movement of the legs or opisthotonos. With this method the traumatic complications of the unmodified electro-convulsion are avoided; there is no serious rise in blood pressure; the post-convulsive excitement and the patient's discomfort are minimized. This method can be safely used in cases in which unmodified convulsion therapy would be dangerous. In the series of cases reported there was no significant effect of the drugs on the electrical convulsion threshold; and the results of treatment were similar to those obtained with unmodified electro-convulsion therapy.

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Comment

The shock therapies "accelerate the recovery," in the sense similar to the acceleration of a chemical reaction by heat. In selected cases the period of illness is shortened, economic cost and loss are lessened, and patients can be socialized in a satisfactory fashion. In some instances their usual routine need not be interrupted.

Numerous attempts have been made to reduce the complications of unusual muscular contractions. The above method represents another attempt to provide the utmost safety and eliminate the chief hazard of unmodified convulsion therapy—H.R.M.

Cerebellar Medulloblastoma in Adults

E. B. Spitz and associates of the Hospital of the University of Pennsylvania (*Archives of Neurology and Psychiatry* 57:417, April 1947) report on a study of 30 cases of cerebellar medulloblastoma in patients sixteen years of age and over. In 15 of these cases the tumor was in one cerebellar hemisphere; in 12 it was limited to the vermis; and in 3 it had evidently originated in the vermis but also involved one of the cerebellar hemispheres. In 21 cases the tumor was soft and widely infiltrating, in 9 cases "fairly" discrete and somewhat firmer. Microscopically these tumors were the same

as the tumors in a larger series of medulloblastoma in children. Signs of acute progressing increased intracranial pressure were the first symptoms in the great majority of these cases. Operation was done to restore the circulation of the cerebrospinal fluid by unblocking the aqueduct and the fourth ventricle, which often required removal of large amounts of tumor. This was followed by radiation therapy according to the method employed by the Department of Radiology of the Hospital. Repeated ventriculoencephalograms were made, which in some cases made it possible to treat recurrent or seeding tumors before they caused clinical symptoms. Two of the patients in this series were not adequately followed up, but both are known to have lived at least a year after operation; one patient died before operation was done and 6 died within thirty days after operation. Of the 21 patients adequately followed up, 13 lived for three years or more, 9 lived for four years or more and 5, or 24 per cent, lived for five years or more after operation; 3 patients are still living, three, six and eight years respectively after operation. Seeding occurred in 38 per cent of these 21 patients. In the larger series of medulloblastoma in children under sixteen years of age, none of the patients lived more than three and a half years after operation. No relation could be found between the gross or histologic characteristics of the tumor and the period of survival.

COMMENT

This paper is of value in directing the attention of clinicians to the possibility of the occurrence of such a tumor in adults. While the future of these cases is notably bad, the reviewer was much impressed with the survival period of some of these patients, particularly in the older age group.

The tendency of this tumor to seed proves the necessity for early diagnosis and prompt surgical attention, if even then this distressing end result can be avoided. Recently Polmeeteer and Kernohan in a study of meningeal gliomatosis showed that 20 of 42 cases arose from the medulloblastoma.

This highly malignant tumor can not be regarded as curable in any case, and at the present time because of the ominous future little satisfaction is gained in making the diagnosis.—H.R.M.

Meningeal Reaction with Abscess of the Brain

B. J. Alpers and F. M. Forster (*Archives of Neurology and Psychiatry* 57:307, March 1947) report histopathologic studies of the meninges in 19 cases with 42 brain abscesses; there was no case of generalized purulent meningitis in the series. In all but one instance there was some involvement of the meninges adjacent to or in the neighborhood of the abscess. This focal meningitis varied from acute purulent to chronic adhesive meningitis with various intermediary stages between these two extreme types. In the cases in which the abscess closely approached or even involved the pia, the meninges near the abscess showed acute inflammatory changes. In the 6 cases of acute brain abscess with no evidence of encapsulation, the meningeal changes were of the acute inflammatory type, but in one of these cases there was evidence that the meningitis was subsiding. In the cases of well encapsulated abscess, with one exception, the meningeal changes were of the chronic type. In 13 cases the meninges at a distance from the abscess were normal in every respect showing no cellular infiltration; in 6 cases there was some cellular infiltration of the meninges at a distance from the abscess. In all 6 cases there was a moderate amount of lymphocytic infiltration and in 3 cases a few polymorphonuclear cells were also present. These studies clearly showed that the meningeal reaction associated with brain abscess is in the beginning a localized process, and that as a rule the meningeal reaction is of such a nature that it prevents the spread of the process to adjacent areas. In this series spinal fluid studies were available in only 7 cases so that no exact correlation can be made between the acuteness of the abscess and the spinal fluid findings, but in general it may be stated that the cell count of the spinal fluid is higher with an acute abscess and lower with a chronic abscess. A low lymphocytic pleocytosis should indicate a chronic abscess, but if the limiting process in the meningeal reaction tends to prevent the entrance of cells into the spinal fluid, the process may be more acute than is indicated by the cell

count; a polymorphonuclear pleocytosis, however, indicates an acute abscess that has not encapsulated.

COMMENT

The above study is one of significance. The authors have been fortunate in the varied nature and amount of their pathologic material studies. It is important to observe their conclusions even in this chemotherapeutic era of penicillin and sulfonamides. In general their observations confirm the experience of practical clinicians in the handling of suspected brain abscesses.

This reviewer has been impressed with the thrombophlebitis factor leading to the acute encephalitic process or brain abscess. It is not always necessary to perform an analysis of the cerebrospinal fluid for the history and physical findings provide the essential data in many instances.—H.R.M.

Relation of Acute Mucosal Hemorrhages and Ulcers of Gastrointestinal Tract to Intracranial Lesions

G. S. Strassmann *(Archives of Neurology and Psychiatry* 57:145, Feb 1947) reports a study of gastrointestinal lesions in 196 autopsies in patients from two state hospitals for mental diseases. In 59 cases (30.1 per cent) no mucosal hemorrhage was found in the gastrointestinal tract; severe cerebrovascular lesions were present in only 3 of these cases. In 89 cases mucosal hemorrhages were found in the stomach or duodenum, often associated with hemorrhages in other parts of the intestinal tract; there were 6 cases in this group in which there was a severe cerebral lesion. In 24 cases there were mucosal hemorrhages of the stomach or duodenum with brownish fluid in stomach and tarry stools; in 5 of these cases local circulatory disturbances may have been the cause of the gastrointestinal bleeding; in 9 of the other cases there were gross intracranial lesions. In 14 of the cases in this series acute hemorrhagic ulcerations were found in the upper gastrointestinal tract, lower esophagus, stomach or upper duodenum; similar lesions were found in 12 cases from the Office of the Chief Medical Examiner of New York City; in 30 cases from this series and from the Office of the Chief Medical Examiner, there were softening and perforation of the esophagus, stomach or duodenum. In all but 2 of these cases there were intracranial lesions of various types. The observed association of the gastrointestinal lesions and the intracranial lesions in this series and also reported by others suggests that various intracranial lesions stimulate the autonomic centers and that the irritation thus produced may result in mucosal hemorrhage, ulcerations or softenings of the gastrointestinal tract or a combination of such lesions.

COMMENT

Since Harvey Cushing published his experiences with the occurrence of ulcerations of the gastro-intestinal tract in brain tumor cases, clinicians with diversified interests have paid increasing attention to this association of mucosal ulceration and cerebral states. The observations contained in this abstracted article tend to confirm the experiences of this commentator when post-mortem studies have been made in instances of cerebral hemorrhage.—H.R.M.

OPHTHALMOLOGY

Ophthalmoscopic Changes Associated with Hypertensive Vascular Disease as a Guide to Sympathectomy

M. Cohen *(Archives of Ophthalmology* 37:491, April 1947) reports the examination of the fundi of 90 patients with essential hypertension before and after thoracolumbar sympathectomy. In 23 of these patients the fundi were normal at the first examination; one of these patients refused sympathectomy. The fundi continued to be normal in the remaining 22 patients; the high diastolic pressure was reduced in all but 2 of these patients within six months after sympathectomy. In 43 patients the changes in the fundi before sympathectomy were classified as "mild." There was usually a contraction of some arterioles with prominence of the

central light reflex; the retinal veins were moderately engorged and tortuous with compression at some arteriolar crossings; there were a few small hemorrhages and exudates. One of these patients refused operation. In 10 the fundi, which had shown arteriolar spasm, became normal after sympathectomy; in 32 signs of arteriolar sclerosis persisted, but edema, hemorrhages and exudates disappeared. One of the patients in this group died of coronary disease two days after operation; all but 5 of the other patients showed a marked reduction of diastolic pressure six months after sympathectomy. In 20 patients the first ophthalmoscopic examination showed lesions of the fundi of moderate degree with a silvery white central light reflex of the retinal arterioles, congestion, local compression and deflection of some venules, slight edema of the disk, areas of edema in the retina, diffuse hemorrhages and exudates. In this group one patient refused operation, and operation was not done in another case because of organic disease. In 2 patients with preoperative arteriolar spasm, the fundi became normal after sympathectomy; in 16 there was a residue of arteriolar sclerosis but the edema, hemorrhages and exudates disappeared, although in 3 cases fresh hemorrhages and exudates appeared temporarily within two days after operation. In all but 5 of these patients the diastolic blood pressure was reduced six months after sympathectomy; of 2 patients whose blood pressure was not reduced, 1 died of coronary disease four months after sympathectomy and one eight months after operation. In 4 patients the lesions of the fundi were severe with massive areas of edema of the retina, profuse hemorrhages, thromboses, exudates and star-shaped figure in the mascular area. Operation was not done in 2 of these cases because of concomitant organic disease. One patient died of tumor of the adrenal shortly after operation and one of coronary occlusion a month after operation. While the advisability of sympathectomy in any case of essential hypertension is decided primarily by the internist, the ophthalmoscopic examination is of definite aid, because the fundic lesions reflect very closely the progression of the clinical manifestations of the disease. The association of retinal arteriolar sclerosis with hypertension is significant and requires further investigation in relation to prognosis.

Comment

The effect of sympathectomy in hypertension is often amazing. The chorioretinal changes are most favorably influenced and it is difficult to explain why.—R.I.L.

Penicillin Treatment of Eyelid Infections

C. A. Noe *(American Journal of Ophthalmology* 50:477, April 1947) reports the use of penicillin locally in the treatment of 55 cases of acute and chronic infections of the eyelid and conjunctiva. Both drops (2,500 units of penicillin per cc.) and an ointment (1,000 units per gm.) were used. The ointment was prepared by dissolving penicillin in a small amount of distilled water and mixing it with amber petrolatum. Drops were used for the treatment of conjunctivitis, being instilled every hour during the day and once or twice during the night. The ointment was used in blepharitis, applied to the lid margins three times a day. In blepharoconjunctivitis a combination of the drops and ointment was employed. In several cases of acute meibomitis, the ointment was used, and in addition hot compresses were applied for twenty minutes three times a day. Of 3 cases of chronic blepharitis, one was markedly improved in three weeks; the other 2 cases showed no improvement. Of 17 cases of chronic conjunctivitis, 8 were cured within a week and 3 in two weeks; 4 cases showed improvement in two weeks and 2 no improvement. Of 10 cases of acute conjunctivitis, 6 were cured in a week, and 2 were cured in two weeks; one improved in two weeks and one showed no improvement. Of 14 cases of recurrent blepharitis and conjunctivitis, 3 were cured in two weeks; 4 were improved in one week; all recurred later but again showed improvement under penicillin treatment; 5 cases were improved in two weeks; 2 showed no improvement. Two of 5 cases of chronic blepharitis and

conjunctivitis were cured in one week; 2 were improved in two weeks and one showed no improvement. Of 6 cases with acute meibomitis, 4 were cured in one week and 2 were improved in two weeks. Nine of the 55 patients treated with penicillin developed an allergic dermatitis of the lids, which cleared up when penicillin was stopped. Cultures were made in all cases except the acute meibomitis cases; in some of the cases that were cured, cultures were negative; while some of the cured cases showed hemolytic *Staphylococcus aureus,* several cases showing this same organism were not cured. While the results of penicillin treatment in the group as a whole were not better than with other methods of treatment, there were some individual cases in which the effects of penicillin therapy were "remarkable."

COMMENT

Penicillin is an important remedy for local treatment of infections of the lids and corneal ulcers. The type of case most favorably influenced has not been worked out yet. The sulfonamides also have done much for certain cases but, up to the present, they are a trial and error proposition.—R.I.L.

Use of Frozen-Dried Cornea as Transplant Material

I. H. Leopold and F. H. Adler (*Archives of Ophthalmology,* 37:268, March 1947) report experiments on rabbits in which frozen-dried rabbit cornea was transplanted to normal rabbit eyes. These experiments were undertaken because at the present time the demand for donor corneas exceeds the supply. With the present methods of preservation corneal tissue that has been kept for over seventy-two hours has been found to be unsatisfactory for transplantation. In the authors' experiments the corneal tissue was removed from rabbits' eyes, frozen and dried by a process similar to that used for the preservation of nerves. The frozen dried corneal tissue was rehydrated before being used as a transplant. This rehydrated corneal tissue was used as a transplant after excising the recipient cornea in 75 rabbit eyes. In 59 cases, the transplant healed in the recipient eye, and in 19 cases corneal vascularization occurred. Infection was an early complication in 6 of the 59 takes, and in 9 corneal edema occurred. The transplants caused no unusual host reaction, but none were transparent at any time during a six months' period of observation. In 20 rabbits' eyes in which fresh corneal transplants were used, the graft healed in place and remained transparent in 7 cases; the transplant failed to take in only 2 cases, but did not become transparent. The rehydrated preserved corneal tissue remained opalescent or bluish gray, whatever rehydrating solution was employed. This indicates that there was some change in the preserved corneal tissue that interfered with normal returgescence, although not with a normal take.

COMMENT

Corneal transplant is a major accomplishment of ophthalmic surgery. Every case improved by this procedure is one more patient removed from the blind group. It was formerly thought essential to make the transfer from the donor eye to the patient immediately. The plan of establishing eyebanks in large centers widens the possibilities for those in need but living in smaller communities. Just how long the eye from whch the graft is taken may be kept before operating is in question but the scientific investigation going on will eventually setile many questions inevitably associated with a new and very technical procedure.—R.I.L.

Nutritional Retrobulbar Neuritis

F. D. Carroll (*American Journal of Ophthalmology,* 30:172, February 1947) reports a study of a type of retrobulbar neuritis that has been observed among children in Jamaica, British West Indies, for many years. In the cases studied the visual acuity varied from 20/70 to 20/200 when the patient was first seen. Early in the disease the optic discs were normal, but later showed marked temporal pallor; this pallor was sometimes seen to persist in children who had completely recovered their vision with no visual field defects, under treatment. Visual fields were diffi-

cult to determine in the younger children, but in 6 of 12 older children studied, definite central or caeco-central scotomas, usually small, were obtained. Approximately 10 per cent of the children with retrobulbar neuritis also showed partial nerve deafness; and the majority showed other symptoms of vitamin-B-complex deficiency. These patients were treated usually with brewer's yeast and efforts made to improve the general diet. Of 72 children treated, 42, or 58 per cent, recovered or were markedly improved. If, however, a good diet is not continued, the condition may recur. The author has recently seen a case in a man twenty-four years of age, who lived in Jamaica all his life, and noted blurred vision and impaired hearing at the age of eleven years. Examination of this patient showed vision of 20/200 in both eyes; marked temporal pallor; caeco-central scotomas and normal peripheral fields. Hearing tests showed a peculiar type of nerve deafness. However, at the time of this examination, the patient showed no other signs of nutritional deficiency. The retrobulbar neuritis observed in children in Jamaica closely resembles that observed in American soldiers who were Japanese prisoners-of-war. These men had no adequate food for prolonged periods and developed multiple deficiency diseases, usually within two or three months. At the same time, decreased vision was noted. If the diet was improved, the vision often improved. The author examined a number of these patients after their return to the United States. The vision ranged from 20/70 to 20/200; there was definite temporal pallor of the optic discs in some cases, in others the pallor was slight. In all cases there was a small pericentral or caeco-central scotoma with normal peripheral fields. When an adequate diet was given with the addition of yeast, whi contains all the vitamin-B fractions, the vision improved. The diet of these patients during their imprisonment was deficient in many essentials and it is difficult to determine what factors were responsible for the visual loss.

Comment

Retrobulbar neuritis was observed scientifically in the decade after the Civil War. The first reports came from Albrecht von Graefe and his uncle, Albert von Graefe. It was called retinal ischemia because of the shrunken arteries in the retinas of two children suffering probably from poisoning by worm powders. Chronic cases showed atrophy of the papello-macular bundle in the nerve stem, hence the name. Not many years ago, the effects of wood alcohol were shown in the ganglion cells of the retina and in the cells of the optic thalamus. It is not improbable that the lesions in the nerve stem are secondary to poisoning of the ganglion cells. The same group of symptoms is a part of multiple sclerosis and the hereditary form of optic atrophy. It is not surprising to hear of an endemic type limited to a locality in the tropics or semi-tropics. Beriberi is localized but is associated with dietary deficiencies. Eventually virus infection may be demonstrated as a factor in retrobulbar neuritis.—R.I.L.

UROLOGY

Gelfoam Hemostasis in Prostatic Surgery

S. A. MacDonald and R. E. Powell (*Journal of Urology*, 57:812, May 1947) report the use of Gelfoam in prostatectomy. Gelfoam closely resembles fibrin foam and is used in the same way. Before use it is soaked in thrombin solution, in which it must be kneaded to expel air from the pores; this causes shrinkage, but the Gelfoam becomes soft and pliable. It can be handled easily and cut or molded to fit any cavity without breaking or crumbling. It should be prepared for use during the setting-up stage of the operation. It is best suited for use in one-stage prostatectomies in which a suprapubic trocar and catheter have been used for preliminary drainage. After removal of the prostate, the fossa is examined for arterial bleeding; if large arteries are found to be the source of bleeding, they should be clamped and ligated or coagulated. The fossa is then filled with Gelfoam; it has been found best to place

count; a polymorphonuclear pleocytosis, however, indicates an acute abscess that has not encapsulated.

COMMENT

The above study is one of significance. The authors have been fortunate in the varied nature and amount of their pathologic material studies. It is important to observe their conclusions even in this chemotherapeutic era of penicillin and sulfonamides. In general their observations confirm the experience of practical clinicians in the handling of suspected brain abscesses.

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G. S. Strassmann *(Archives of Neurology and Psychiatry* 57:145, Feb 1947) reports a study of gastrointestinal lesions in 196 autopsies in patients from two state hospitals for mental diseases. In 59 cases (30.1 per cent) no mucosal hemorrhage was found in the gastrointestinal tract; severe cerebrovascular lesions were present in only 3 of these cases. In 89 cases mucosal hemorrhages were found in the stomach or duodenum, often associated with hemorrhages in other parts of the intestinal tract; there were 6 cases in this group in which there was a severe cerebral lesion. In 24 cases there were mucosal hemorrhages of the stomach or duodenum with brownish fluid in stomach and tarry stools; in 5 of these cases local circulatory disturbances may have been the cause of the gastrointestinal bleeding; in 9 of the other cases there were gross intracranial lesions. In 14 of the cases in this series acute hemorrhagic ulcerations were found in the upper gastrointestinal tract, lower esophagus, stomach or upper duodenum; similar lesions were found in 12 cases from the Office of the Chief Medical Examiner of New York City; in 30 cases from this series and from the Office of the Chief Medical Examiner, there were softening and perforation of the esophagus, stomach or duodenum. In all but 2 of these cases there were intracranial lesions of various types. The observed association of the gastrointestinal lesions and the intracranial lesions in this series and also reported by others suggests that various intracranial lesions stimulate the autonomic centers and that the irritation thus produced may result in mucosal hemorrhage, ulcerations or softenings of the gastrointestinal tract or a combination of such lesions.

COMMENT

Since Harvey Cushing published his experiences with the occurrence of ulcerations of the gastro-intestinal tract in brain tumor cases, clinicians with diversified interests have paid increasing attention to this association of mucosal ulceration and cerebral states. The observations contained in this abstracted article tend to confirm the experiences of this commentator when post-mortem studies have been made in instances of cerebral hemorrhage.—H.R.M.

OPHTHALMOLOGY

Ophthalmoscopic Changes Associated with Hypertensive Vascular Disease as a Guide to Sympathectomy

M. Cohen *(Archives of Ophthalmology* 37:491, April 1947) reports the examination of the fundi of 90 patients with essential hypertension before and after thoracolumbar sympathectomy. In 23 of these patients the fundi were normal at the first examination; one of these patients refused sympathectomy. The fundi continued to be normal in the remaining 22 patients; the high diastolic pressure was reduced in all but 2 of these patients within six months after sympathectomy. In 43 patients the changes in the fundi before sympathectomy were classified as "mild." There was usually a contraction of some arterioles with prominence of the

central light reflex; the retinal veins were moderately engorged and tortuous with compression at some arteriolar crossings; there were a few small hemorrhages and exudates. One of these patients refused operation. In 10 the fundi, which had shown arteriolar spasm, became normal after sympathectomy; in 32 signs of arteriolar sclerosis persisted, but edema, hemorrhages and exudates disappeared. One of the patients in this group died of coronary disease two days after operation; all but 5 of the other patients showed a marked reduction of diastolic pressure six months after sympathectomy. In 20 patients the first ophthalmoscopic examination showed lesions of the fundi of moderate degree with a silvery white central light reflex of the retinal arterioles, congestion, local compression and deflection of some venules, slight edema of the disk, areas of edema in the retina, diffuse hemorrhages and exudates. In this group one patient refused operation, and operation was not done in another case because of organic disease. In 2 patients with preoperative arteriolar spasm, the fundi became normal after sympathectomy; in 16 there was a residue of arteriolar sclerosis but the edema, hemorrhages and exudates disappeared, although in 3 cases fresh hemorrhages and exudates appeared temporarily within two days after operation. In all but 5 of these patients the diastolic blood pressure was reduced six months after sympathectomy; of 2 patients whose blood pressure was not reduced, 1 died of coronary disease four months after sympathectomy and one eight months after operation. In 4 patients the lesions of the fundi were severe with massive areas of edema of the retina, profuse hemorrhages, thromboses, exudates and star-shaped figure in the mascular area. Operation was not done in 2 of these cases because of concomitant organic disease. One patient died of tumor of the adrenal shortly after operation and one of coronary occlusion a month after operation. While the advisability of sympathectomy in any case of essential hypertension is decided primarily by the internist, the ophthalmoscopic examination is of definite aid, because the fundic lesions reflect very closely the progression of the clinical manifestations of the disease. The association of retinal arteriolar sclerosis with hypertension is significant and requires further investigation in relation to prognosis.

COMMENT

The effect of sympathectomy in hypertension is often amazing. The chorioretinal changes are most favorably influenced and it is difficult to explain why.—R.I.L.

Penicillin Treatment of Eyelid Infections

C. A. Noe (*American Journal of Ophthalmology* 50:477, April 1947) reports the use of penicillin locally in the treatment of 55 cases of acute and chronic infections of the eyelid and conjunctiva. Both drops (2,500 units of penicillin per cc.) and an ointment (1,000 units per gm.) were used. The ointment was prepared by dissolving penicillin in a small amount of distilled water and mixing it with amber petrolatum. Drops were used for the treatment of conjunctivitis, being instilled every hour during the day and once or twice during the night. The ointment was used in blepharitis, applied to the lid margins three times a day. In blepharoconjunctivitis a combination of the drops and ointment was employed. In several cases of acute meibomitis, the ointment was used, and in addition hot compresses were applied for twenty minutes three times a day. Of 3 cases of chronic blepharitis, one was markedly improved in three weeks; the other 2 cases showed no improvement. Of 17 cases of chronic conjunctivitis, 8 were cured within a week and 3 in two weeks; 4 cases showed improvement in two weeks and 2 no improvement. Of 10 cases of acute conjunctivitis, 6 were cured in a week, and 2 were cured in two weeks; one improved in two weeks and one showed no improvement. Of 14 cases of recurrent blepharitis and conjunctivitis, 3 were cured in two weeks; 4 were improved in one week; all recurred later but again showed improvement under penicillin treatment; 5 cases were improved in two weeks; 2 showed no improvement. Two of 5 cases of chronic blepharitis and

conjunctivitis were cured in one week; 2 were improved in two weeks and one showed no improvement. Of 6 cases with acute meibomitis, 4 were cured in one week and 2 were improved in two weeks. Nine of the 55 patients treated with penicillin developed an allergic dermatitis of the lids, which cleared up when penicillin was stopped. Cultures were made in all cases except the acute meibomitis cases; in some of the cases that were cured, cultures were negative; while some of the cured cases showed hemolytic *Staphylococcus aureus,* several cases showing this same organism were not cured. While the results of penicillin treatment in the group as a whole were not better than with other methods of treatment, there were some individual cases in which the effects of penicillin therapy were "remarkable."

COMMENT

Penicillin is an important remedy for local treatment of infections of the lids and corneal ulcers. The type of case most favorably influenced has not been worked out yet. The sulfonamides also have done much for certain cases but, up to the present, they are a trial and error proposition.—R.I.L.

Use of Frozen-Dried Cornea as Transplant Material

I. H. Leopold and F. H. Adler (*Archives of Ophthalmology,* 37:268, March 1947) report experiments on rabbits in which frozen-dried rabbit cornea was transplanted to normal rabbit eyes. These experiments were undertaken because at the present time the demand for donor corneas exceeds the supply. With the present methods of preservation corneal tissue that has been kept for over seventy-two hours has been found to be unsatisfactory for transplantation. In the authors' experiments the corneal tissue was removed from rabbits' eyes, frozen and dried by a process similar to that used for the preservation of nerves. The frozen dried corneal tissue was rehydrated before being used as a transplant. This rehydrated corneal tissue was used as a transplant after excising the recipient cornea in 75 rabbit eyes. In 59 cases, the transplant healed in the recipient eye, and in 19 cases corneal vascularization occurred. Infection was an early complication in 6 of the 59 takes, and in 9 corneal edema occurred. The transplants caused no unusual host reaction, but none were transparent at any time during a six months' period of observation. In 20 rabbits' eyes in which fresh corneal transplants were used, the graft healed in place and remained transparent in 7 cases; the transplant failed to take in only 2 cases, but did not become transparent. The rehydrated preserved corneal tissue remained opalescent or bluish gray, whatever rehydrating solution was employed. This indicates that there was some change in the preserved corneal tissue that interfered with normal returgescence, although not with a normal take.

COMMENT

Corneal transplant is a major accomplishment of ophthalmic surgery. Every case improved by this procedure is one more patient removed from the blind group. It was formerly thought essential to make the transfer from the donor eye to the patient immediately. The plan of establishing eyebanks in large centers widens the possibilities for those in need but living in smaller communities. Just how long the eye from whch the graft is taken may be kept before operating is in question but the scientific investigation going on will eventually settle many questions inevitably associated with a new and very technical procedure.—R.I.L.

Nutritional Retrobulbar Neuritis

F. D. Carroll (*American Journal of Ophthalmology,* 30:172, February 1947) reports a study of a type of retrobulbar neuritis that has been observed among children in Jamaica, British West Indies, for many years. In the cases studied the visual acuity varied from 20/70 to 20/200 when the patient was first seen. Early in the disease the optic discs were normal, but later showed marked temporal pallor; this pallor was sometimes seen to persist in children who had completely recovered their vision with no visual field defects, under treatment. Visual fields were diffi-

cult to determine in the younger children, but in 6 of 12 older children studied, definite central or caeco-central scotomas, usually small, were obtained. Approximately 10 per cent of the children with retrobulbar neuritis also showed partial nerve deafness; and the majority showed other symptoms of vitamin-B-complex deficiency. These patients were treated usually with brewer's yeast and efforts made to improve the general diet. Of 72 children treated, 42, or 58 per cent, recovered or were markedly improved. If, however, a good diet is not continued, the condition may recur. The author has recently seen a case in a man twenty-four years of age, who lived in Jamaica all his life, and noted blurred vision and impaired hearing at the age of eleven years. Examination of this patient showed vision of 20/200 in both eyes; marked temporal pallor; caeco-central scotomas and normal peripheral fields. Hearing tests showed a peculiar type of nerve deafness. However, at the time of this examination, the patient showed no other signs of nutritional deficiency. The retrobulbar neuritis observed in children in Jamaica closely resembles that observed in American soldiers who were Japanese prisoners-of-war. These men had no adequate food for prolonged periods and developed multiple deficiency diseases, usually within two or three months. At the same time, decreased vision was noted. If the diet was improved, the vision often improved. The author examined a number of these patients after their return to the United States. The vision ranged from 20/70 to 20/200; there was definite temporal pallor of the optic discs in some cases, in others the pallor was slight. In all cases there was a small pericentral or caeco-central scotoma with normal peripheral fields. When an adequate diet was given with the addition of yeast, whi contains all the vitamin-B fractions, the vision improved. The diet of these patients during their imprisonment was deficient in many essentials and it is difficult to determine what factors were responsible for the visual loss.

Comment

Retrobulbar neuritis was observed scientifically in the decade after the Civil War. The first reports came from Albrecht von Graefe and his uncle, Albert von Graefe. It was called retinal ischemia because of the shrunken arteries in the retinas of two children suffering probably from poisoning by worm powders. Chronic cases showed atrophy of the papellomacular bundle in the nerve stem, hence the name. Not many years ago, the effects of wood alcohol were shown in the ganglion cells of the retina and in the cells of the optic thalamus. It is not improbable that the lesions in the nerve stem are secondary to poisoning of the ganglion cells. The same group of symptoms is a part of multiple sclerosis and the hereditary form of optic atrophy. It is not surprising to hear of an endemic type limited to a locality in the tropics or semi-tropics. Beriberi is localized but is associated with dietary deficiencies. Eventually virus infection may be demonstrated as a factor in retrobulbar neuritis.—R.I.L.

UROLOGY

Gelfoam Hemostasis in Prostatic Surgery

S. A. MacDonald and R. E. Powell (*Journal of Urology*, 57:812, May 1947) report the use of Gelfoam in prostatectomy. Gelfoam closely resembles fibrin foam and is used in the same way. Before use it is soaked in thrombin solution, in which it must be kneaded to expel air from the pores; this causes shrinkage, but the Gelfoam becomes soft and pliable. It can be handled easily and cut or molded to fit any cavity without breaking or crumbling. It should be prepared for use during the setting-up stage of the operation. It is best suited for use in one-stage prostatectomies in which a suprapubic trocar and catheter have been used for preliminary drainage. After removal of the prostate, the fossa is examined for arterial bleeding; if large arteries are found to be the source of bleeding, they should be clamped and ligated or coagulated. The fossa is then filled with Gelfoam; it has been found best to place

four or five fragments of Gelfoam in position and apply suction until the cavity is filled completely. If the adenoma removed is large, from twenty to thirty pieces of Gelfoam may be required. Small caliber straight tubes fastened high in the fundus have been found best for drainage; no bladder irrigations are employed postoperatively but simple bed-side or suction drainage. In 15 cases of prostatectomy fibrin foam was used in 3 cases, Gelfoam in the remainder. There was no difference in the hemostatic effect of the two; the Gelfoam was somewhat easier to handle. There was only one case in which satisfactory hemostatis was not obtained, and this was early in the series. In the other cases hemostasis was almost complete; the drainage fluid was never more than slightly pink and became entirely clear in forty-eight to seventy-two hours; no clots formed in any case. The series of cases reported is small, but the authors consider that the results indicate the value of this new method of hemostasis in prostatic surgery.

COMMENT

If in Gelfoam we have a hemostatic which will prevent postoperative bleeding, early or late, we have certainly advanced far on the road of making prostatic surgery safe. Time will tell because so many things, devices and preparations have been tried, had their day and passed out.

V.C.P.

The Composition of Urinary Calculi

C. L. Prien and C. Frondel *(Journal of Urology,* 57:949, June 1947) report a study of the composition of urinary calculi by means of optical and x-ray crystallography, i.e., examination by polarized light and by x-ray diffraction photography. Chemical analysis of urinary calculi has been found to be unsatisfactory as compared with these modern physical methods of identification. All urinary calculi, except for the very rare fibrin and bacterial calculi, are crystalline and therefore amenable to analysis by these physical methods. The crystalline components of urinary calculi, as found by these methods, are calcium oxalate monohydrate (whewellite), calcium oxalate dehydrate (weddellite), magnesium ammonium phosphate hexahydrate (stuvite), carbonate-apatite and hydroxyl-apatite (collectively called apatite), calcium hydrogen phosphate dehydrate (Lushite), uric acid, cystine and sodium acid urate. In a series of approximately 700 calculi, calcium carbonate, cholesterol xanthine and indigo were not found. In this series of calculi, 36.1 per cent were pure calcium oxalate; 31 per cent mixed calcium oxalate-apatite; these two groups, therefore, made up 67.1 per cent of the total. These calculi were usually found in acid, sterile urine. Pure magnesium ammonium phosphate hexahydrate, pure apatite and mixed magnesium ammonium phosphate hexahydrate-apatite calculi made up 19.5 per cent of the total; these calculi were usually found in alkaline, infected urine. Uric acid and cystine were found more often in mixed than in pure form; sodium acid urate was found in only one instance and was the only urate found in this series. The authors are of the opinion that the physical methods of analysis used in this study (optical and x-ray crystallography) "provide powerful tools" for the study of various aspects of urinary calculi.

COMMENT

The most interesting feature of this study brings out the fact that infection need not be present for the formation of stones. In acid sterile urine occur 36.1 per cent pure calcium oxalate, and 31 per cent mixed calcium oxalate-apatite, making 67.1 per cent of the whole, but were these urines cultured to determine the presence or absence of infection? The article omits that all-important point. The other stones appear to occur only in infected urine.

V.C.P.

Conservative Treatment of Renal and Ureteral Colic

J. H. Morrissey *(Journal of Urology,* 57:635, April 1947) notes that with modern chemotherapy to combat infection there is a tendency to treat ureteral calculi more conservatively. This has increased the need for medication that will give symptomatic relief and possibly promote spontaneous passage of the calculus by controlling spasm. The author has employed Donnatal, a spasmolytic but non-narcotic

preparation in the treatment of cases of renal and ureteral colic due to calculus, cases with renal and ureteral colic in which no calculus could be demonstrated, and in cases of bladder neck spasm, Donnatal has also been employed to prevent and relieve post-cystoscopic reactions. In cases of renal and ureteral colic and bladder neck spasm, 2 tablets of Donnatal were given following the onset of symptoms, followed by another 2 tablets in two hours, and then by one tablet every four hours. If the pain did not subside or became worse over a thirty-six hour period, Pantopon or Demerol was given by mouth or subcutaneously. In a series of 37 cases of renal and ureteral colic due to calculus and 69 cases with similar symptoms in which no calculus was found, Donnatal relieved symptoms without the use of Pantopon or Demerol in all but 10 of the cases of calculus, and in all of the cases without calculus; in most of these cases symptoms were relieved in thirty-six hours. In 45 cases of bladder neck spasm, Donnatal also gave good results; this group included 9 cases of stricture of the urethra and chronic granular urethrism in women in which Donnatal was given for forty-eight hours postoperatively. In 197 cases in which Donnatal was used to relieve discomfort and spasm following cystoscopy and pyelography, 2 tablets were given two hours before cystoscopy and 2 more tablets at the beginning of the procedure. After the procedure, including pyelography, was completed 1 tablet of Donnatal was given every three hours; and the patient was instructed to drink water as copiously as possible. With this method less than 10 per cent of the patients required Pantopon or Demerol for relief of post-cystoscopic pain and spasm. In over 75 per cent of these cases a 16 Wappler cystoscope was used, which causes less trauma and is less irritating than larger calibre cystoscopes often employed; the size of the ureteral catheters, however, is the same as with other cystoscopes and the so-called post-cystoscopic reactions are usually of renal and ureteral origin, as shown by the type of referred pain and the distribution and location of the spasm.

COMMENT

It is interesting to note the rather large number of various antipasmodics and sedatives now employed in urology and their repeated and distributive dosages. One old combination cannot be denied. I have used it ever since my New York Hospital days where the honored and late Dr. Robert F. Weir taught me to. I have never seen it fail to abort or check a urethral chill or to allay spasm due to whatever cause: Fluid Extract of Aconite m. 1, Sulfate of Quinine grs. 3 to 5; Sulfate of Morphine gr. 1/8 to 1/4. In converting this to tablet form I substituted for the Fluid Extract of Aconite a solid preparation, namely, Aconitine gr. 1/200. I can recommend it as efficient and trustworthy. One tablet is given at the end of the procedure and repeated if necessary (which hardly ever happens) in 3 hours. It has the advantage of being composed of standard drugs.

V.C.P.

Streptomycin Treatment of Urinary Tract Infections

H. W. Harris and associates *(American Journal of Medicine,* 2:229, March 1947) report the treatment of 21 cases of urinary tract infection in adults with streptomycin; in all these cases the organisms isolated by culture from the urine were sensitive to streptomycin *in vitro*. In 17 cases, the duration of the urinary tract infection was known; in 10 of these cases it was a year or more; in 2 between six and twelve months; in 4 about two months; and in one only two weeks. The other four patients in this series had chronic infections but the exact duration was not known. In all cases streptomycin was given intramuscularly, the total dosage ranged from 2.5 to 6 gm., given in doses of 0.5 to 1 gm. every four to six hours; in all but 4 patients the daily dosage was 4 gm. or more. An initial intravenous dose of 0.5 to 1 gm. was given in 19 cases before the first intramuscular injection. Streptomycin was continued for about seven days unless some toxic reaction made it necessary to discontinue treatment. In 14 cases, the urine was alkalinized before and during the streptomycin treatment by the oral administration of sodium bicarbonate and potassium citrate; usually 1 gm. of each alkali

was given six times daily, but 4 patients required slightly larger doses, 2 patients were given sodium bicarbonate alone. The alkali therapy was given for a control period of two days or more before beginning streptomycin therapy; in this period there was a significant reduction of bacilluria in only one case. In this series of 21 cases, sterilization of the urine was obtained in 15 cases; in 8 of these the cultures of the urine became negative within twelve hours after the first dose of streptomycin was given; complete and lasting freedom from bacilluria during the period of observation was obtained in only 9 cases. In the 7 cases in which alkalis were not employed, only one patient showed complete and lasting cure of bacilluria, and one other temporary freedom from bacilluria with recurrence in five days. The most serious reaction to streptomycin in this series was an acute labyrinthine disturbance in one patient; 2 other patients had mild vertigo, which occurred only when they stood up. Mild local reactions at the site of injection developed in most cases; in 7 the pain and tenderness were severe enolgh to interfere with ambulation; in 3 other cases, the local reactions was more severe and persisted after treatment was stopped. The results in the series of cases reported indicate that alkalinization of the urine during streptomycin therapy increases the therapeutic effectiveness of streptomycin and reduces the incidence of the development of streptomycin-fastness.

Comment

We have framed the sulfonamides in advantages, disadvantages, limitations and dangers and etched on the glass "Caution Always." In the last 50 years I have seen many urinary antiseptics cross the professional sky and sink beyond the horizon. Of the few which remain streptomycin gives promise of lasting. Even this article, however, admits toxic reaction and discontinuance of treatment. Too little is said about the chronic cases which are the great problem for urologists. It must be remembered that only that is taken into the body which is absorbed by the body, therefore our mucous membranes are like the skin in being a barrier through which medicines pass into the blood stream. If the mucous membrane is damaged beyond certain limits it never recovers be it the intestines or the urinary organs or what not. Hence the grave importance of studying all features of chronic infections. It is a pity that this was not done to the limit in this valuable article.

V.C.P.

PUBLIC HEALTH, INDUSTRIAL MEDICINE AND SOCIAL HYGIENE

Sulfadiazine-Resistant Streptococcal Infections in a Civilian Community

R. D. Johnson and T. L. Hartman *(Journal of Clinical Investigation,* 26:325, March 1947) report a study of the incidence of sulfadiazine-resistant strains of streptococci in a civilian community (Cooperstown, N. Y.) during a small epidemic of scarlet fever. During a program of "mass chemoprophylaxis" of streptococcal infections in the Armed Forces, it was found that the effectiveness of such prophylaxis was lost as soon as sulfonamide-resistant strains appeared among susceptible groups in the Army and Navy. In Cooperstown, the first 8 cases of scarlet fever reported were found to be due to type 19 hemolytic streptococcus, and of these 8 strains were resistant to sodium sulfadiazine in a concentration of 25 mg. per cent. From February 15 to May 1, 1946, streptococcal infections were moderately prevalent in Cooperstown and the surrounding areas, and cultures were sent in to the Otsego County Laboratory. Of these cultures 100 strains were found to belong to group A hemolytic streptococcus. These 100 strains were sent to the Rockefeller Institute for typing and determination of their sulfonamide resistance. The most common type found was

type 3, which was the causative organism in 28 cases. Type 19 was the causative organism in 24 cases, and 23 of these strains were found to be resistant to sodium sulfadiazine (25 mg. per cent). No other strains were resistant to sulfadiazine. Of these cases of sulfonamide-resistant streptococcal infection, 14 were clinical scarlet fever and 9 pharyngitis; otitis media complicated 2 cases (one scarlet fever and one pharyngitis); rheumatic fever followed pharyngitis in one other case. The first patient from whom a type-19 sulfonamide-resistant streptococcus was isolated was a nine year old boy who developed scarlet fever; several of his classmates developed scarlet fever and type 19 sulfonamide-resistant streptococci were isolated from these patients. Most of the other patients showing this type of sulfonamide-resistant hemolytic streptococcus were contacts of these children; however, 3 patients lived in widely separated communities and had no known contact with the other patients. As the type 19 strain of hemolytic streptococcus was known to be a sulfonamide-resistant strain in the Armed Forces, it is probable that this strain was introduced into this civilian community by discharged veterans, some of whom were probably healthy carriers. Since the clinical picture of infections due to sulfonamide-resistant strains of hemolytic streptococcus does not differ from that due to other strains, it is important to determine this property by bacteriologic methods as a guide to the best method of treatment and of prevention of such resistant strain infections.

COMMENT

Sulfonamides cannot be used promiscuously. A number of types of Group A streptococci have been reported in outbreaks as developing resistance. This raises the question as to whether patients with mild infections should be given the drug. Most recover promptly under symptomatic treatment. Fortunately, to date most reports on resistant streptococci usually conclude with the statement that the patients responded well to penicillin, which is always in reserve.

E.G.B.

Inactivation of the Virus of Infectious Hepatitis in Drinking Water

J. R. Neefe and associates *(American Journal of Public Health,* 37:365, April 1947) report a study of methods of inactivation of the virus of infectious hepatitis in drinking water. This study was undertaken because an epidemic of infectious hepatitis had been observed in a summer camp for boys and girls in which the drinking water had been proved to be the means of transmission of the virus (reported in 1945). It was found that coagulation, settling and filtration (diatomite filter) of contaminated water did not entirely eliminate or inactivate the virus, but did cause a prolongation of the incubation period and reduced the incidence of hepatitis by 40 per cent in volunteers drinking the treated water as compared to a control experiment with untreated water. When chlorine was added to such treated water in sufficient amounts to provide total and free residual chlorine concentrations of 0.1 and 0.4 p.p.m. respectively after thirty minutes' contact, this was effective in inactivating the hepatitis virus under the same experimental condition. However, if chlorine in similar concentration was added to water that had not been previously treated by coagulation, settling and filtration, it was not effective in inactivating the hepatitis virus. Further studies of the minimal effective dose of chlorine against the hepatitis virus in coagulated, settled and filtered water are necessary.

COMMENT

A well controlled study confirming one method of transfer of infectious hepatitis. It suggests but does not confirm that many of our water treatment works do not inactivate the virus. This significant point must await further studies on dilution and holding time and their effect on chance contamination. A residual concentration of free chlorine in excess of 0.4 p. p. m. (orthotolidine-arsenite reaction) after 30 minutes would appear from this study to be the determining safety factor.

E.G.B.

Nutritional Factors in Prevention and Treatment of the Hepatic Injury Due to Industrial Poisons

J. H. Eddy, Jr. *(Southern Medical Journal,* 40:171, Feb. 1947) has found that a high protein diet, with inclusion of proteins containing methionine, has a protective value in workers exposed to trinitrotoluene and other hepatotoxins used in industrial processes. Other methods of protection against these toxins should not be neglected. Less toxic agents should be substituted for known hepatotoxins, when possible. Where toxic agents must be used, closed systems should be employed or the operation mechanically ventilated. The use of respirators is unsatisfactory, and is to be recommended only when there is no other method of preventing contact with the toxic agent. All persons who have had hepatitis, who are being treated or have been treated with an arsenical, those with syphilis not under treatment and with gallbladder disease should be excluded from contact with hepatotoxic substances. Rotation of workers exposed to such toxic substances is a safeguard against chronic poisoning. Periodic examination of such workers should be done, and this should include a simple test of the urine for bile. Regulation of the diet of all workers exposed to hepatotoxic substances is an important part of the "health plan" of an industrial establishment. The drinking of skimmed milk is recommended, but other animal proteins are also necessary; in a plant where T.N.T. is handled workers are advised to include in their diet a quart of milk, two eggs, gelatin and cheese daily. All workers who are heavily exposed should be given methionine tablets (3 to 4 gm. daily). The author has also employed methionine in the treatment of cases of hepatitis due to trinitrotoluene poisoning; in 30 cases so treated (reported in 1945), 10 patients were critically ill, but there were no deaths; the average period of hospitalization was thirteen days.

COMMENT

The author summarized his findings of the nutritional value of a high protein diet containing methionine in the prevention and treatment of hepatic injuries due to exposure of the worker to trinitrotoluene and other hepatotoxins used in industry. He also mentioned some of the possible industrial hygiene preventive measures.

Of all points covered by the author, certainly the use of local exhaust ventilation stands out as the most important method of removing the toxic substances from the breathing zone of the worker. The system should be properly designed to remove the contamination below the Maximum Allowable Concentration efficiently, with the least amount of displaced air, since this is of considerable economic importance to a large factory during the heating season. The method of substitution may be possible in some cases, such as the use of toluol for benzol, but in most industrial processes it is not feasible either from an economic or an operational viewpoint.

E.G.B.

A Study of the Hazard of Exposure to Metallic Fumes and Dust in Brass Foundries

W. W. Stalker *(Journal of Industrial Hygiene,* 29:96, March 1947)) reports that in two series of lead and zinc determinations in fumes from 4 brass foundries, a high average lead concentration was found in only one of the 4 foundries in the first series of samples; and the second series showed no high concentration of lead or zinc. In a study of 98 foundrymen from 5 brass foundries, blood and urine analysis showed evidence of excessive lead absorption in a much higher percentage of cases than the stippled red cell counts. These findings indicate that blood and urine analyses of workers in brass foundries give a more accurate indication of total lead exposure than the examination of the blood for stippled red cells or periodic atmospheric analyses. A physical examination of 75 brass foundrymen, including only 25 of those who showed high lead concentration in the urine, indicates that "a mild alimentary type" of lead intoxication may be present in many of these workers. Control of the lead hazard and other fume hazards depends upon general ventilation and special types of fume control and upon the maintenance of a comprehensive medical and sanitation program, requiring the

cooperation of management, workers and the industrial hygienist.

COMMENT

It is interesting to note that analyses of blood and urine from brass foundry workers give a more accurate indication of total lead exposure than periodic atmospheric analyses. Such a finding makes it appear that either periodic atmospheric samples are not representative of average conditions, or that workers subject themselves unnecessarily to fumes by not complying with authorized procedure.

E.G.B.

Universal Serologic Reactivity with Lipid Anticens: Basis for False Positives

R. L. Kahn *(American Journal of Public Health,* 37:293, March 1947) describes universal serological reactions with lipid antigens observed in the laboratory of the University of Michigan Hospital. It has been found that lipid antigen and serum mixtures such as are employed in tests for syphilis can be technically manipulated so that positive precipitation reactions occur in most non-syphilitic persons. In such serum-lipid antigen combinations, low Na Cl concentrations and low temperature are especially favorable for eliciting the precipitation reactions; the precipitates formed are usually dispersible in concentrated Na Cl solution. It is noted also that the same conditions, i.e., low salt concentration and low temperature, are the most favorable for false positive reactions in serodiagnostic tests for syphilis, and that the precipitates formed in such false positive reactions are also usually dispersible in concentrated Na Cl solution. This suggests that there is a close relation between the universal and the false positive reactions; and that the false positives are highly potent universal reactions that "have passed the sero-diagnostic threshold" and have thus been manifested as false positive reactions. In the illustrative case presented, there was a weak false positive reaction but a universal reaction of high potency which evidently reached into the serodiagnostic zone. The serodiagnostic threshold is not the same in all serodiagnostic tests for syphilis. In the more sensitive tests, the serodiagnostic threshold is lower, i.e., more universal reactions will pass this threshold. In developing and modifying serodiagnostic tests for syphilis, therefore, increased specificity rather than increased sensitivity should be emphasized.

COMMENT

A lucid explanation of the occasionally false postive sera and an interesting phenomenon illustrating the necessity of rigid technic in the laboratory. Titers, however, ocassionally as high as 3,700 have been reported and found to be false positives. It remains to be proven that titers of this height with proper technique fall within this explanation or represent a defect in cholesterol metabolism. Rein and Kent in their study of 90 non-syphilitic volunteers (nearly 45,000 tests of all types) observe: "the highest percentage of false positive reactors was observed with the Kahn standard test and the lowest with the Hinton flocculation." (J.A.M.A., Vol. 133, 14:1001).

E.G.B.

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Advance in Treatment of Syphilis

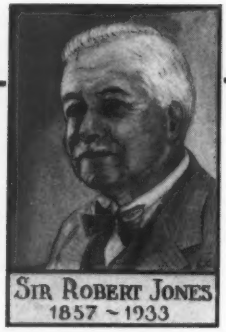
Two weeks of properly given penicillin therapy is probably as effective as two years of weekly injections of drugs in the treatment of syphilis, according to Dr. Evan W. Thomas. Dr. Thomas is associate professor of medicine at the New York University College of Medicine, and director of the Rapid Treatment Center at Bellevue Hospital.

"The advent of quantitative serological tests for syphilis and penicillin for treatment have enormously improved the management of the disease. This does not mean that penicillin is a panacea for syphilis or that we can yet dispense with all previous aids to therapy. But it does mean that we have made tremendous strides in the therapeutic control of this chronic disease."

Medical BOOK NEWS

Edited by
ALFRED E. SHIPLEY, M.D., Dr. P.H.

All books for review and communications concerning Book News should be addressed to the Editor of this department, 1313 Bedford Avenue, Brooklyn 16, N. Y.



Classical Quotations

• Science favours no language, but demands of all its votaries a relentless and faithful search for truth. We have neither time nor desire to wander aimlessly along bypaths, which lead nowhere. Each nation in friendly rivalry is engaged in a struggle to place the art we love upon a sure and indestructible foundation.

SIR ROBERT JONES

Address as First President of the International Society of Orthopaedic Surgery, Paris, October, 1930.

Booklet of English Medicine

The Science & Art of Medicine. By Sir Lionel Whitby, M.D. Cambridge, The University Press (New York, Macmillan Co.), [c. 1946]. 12mo. 24 pages. Paper, 50c.

THE inaugural address of the Regius Professor of Physic at Cambridge defines the relations of the scientific basis and the artistic performance of the practice of medicine. The accent is on chemotherapy, and Sir Lionel is, as he notes, the first of twenty-two Regius Professors since 1540 whose "practice, tastes, training, and original work" have been scientific rather than clinical. Yet he champions the right of the physician to "express his own opinion and practice his own art." This stimulating address shows how much alive the ancient Universities of England are today.

WILLIAM DOCK

Dentistry and Health

Dentistry. An Agency of Health Service. By Malcolm Wallace Carr, D.D.S. New York, Commonwealth Fund, [c. 1946]. 8vo. 219 pages. Cloth $1.50.

This short treatise offers a concise factual picture of a century old profession.

Dr. Carr has chosen a group of experts to collaborate with him in this compact picture of present-day dentistry as a health service. The members of the medical and dental professions will find it absorbingly interesting. It is a must for all in the health teaching field.

VINCENT D. CALLAHAN

Biography

Henry Sewall. Physiologist and Physician. By Gerald B. Webb & Desmond Powell. Baltimore, Johns Hopkins Press, [c. 1946]. 8vo. 191 pages, illustrated. Cloth, $2.75.

Henry Sewall, as a young physiologist, brought medical fame to the University of Michigan by his work on immunization to snake venom. Although he became later a physician, he remained always a physiologist, and his active investigative career was highly symbolic of the change that swept over the world of medicine during the years of his life at the turn of the last century.

J. RAYMOND JOHNSON

Bronchial Asthma

The Diagnosis and Treatment of Bronchial Asthma. By Leslie N. Gay, M.D. Baltimore, Williams & Wilkins, [c. 1946]. 8vo. 334 pages, illustrated. Cloth, $5.00.

This book endeavors to meet the present-day medical needs of the student, internist, or medical consultant. Emphasis is placed upon the value of a comprehensive examination, the proper diagnosis and a thorough history of any patient whose complaint is shortness of breath.

NELSON MILES HOLDEN

Blood

Clinical Hematology. By Maxwell M. Wintrobe, M.D. 2nd Edition. Philadelphia, Lea & Febiger, [c. 1946]. 8vo. 862 pages, illustrated. Cloth, $11.00.

This is a complete text on clinical hematology. Dr. Wintrobe is an authority in this field and his book stands out as a text dealing with hematological problems in a thorough manner. It is highly recommended to students of the subject as well as to general practitioners.

EUGENE R. MARZULLO

Pathology for Nurses

Pathology. By Eugene C. Piette, M.D. 5th Edition. Philadelphia, F. A. Davis Co., [c. 1945]. 8vo. 261 pages, illustrated. Cloth, $2.00.

This brief survey of pathology for nurses is divided into four divisions. The first pertains to the principles of pathology; the second and third units deal with the more specific diseases. The fourth relates to clinical pathology.

The illustrations are rather poorly reproduced.

CASPAR G. BURN

Preventive Medicine

A Future for Preventive Medicine. By Edward J. Stieglitz, M.D. New York, Commonwealth Fund, [c. 1945]. 8vo. 77 pages. Cloth, $1.00.

This is another of a series sponsored by the New York Academy of Medicine. The ideal objectives set forth should stimulate the thinking, especially of the younger generation of physicians by whom the practical application of these ideals must be developed in the not too distant future.

ALFRED E. SHIPLEY

Embryology

Human Embryology. (Prenatal Development of Form and Function). By W. J. Hamilton, M.D., J. D. Boyd, M.D., & H. W. Mossman, Ph.D. Baltimore, Williams & Wilkins Co., [c. 1945]. 8vo. 366 pages, illustrated. Cloth, $7.00.

This book is organized so that there is a continuity of subject matter that is difficult to arrange when separate body systems are being dealt with. The most modern conceptions of development processes based on experimental work performed on lower forms of animal life are discussed.

The book is a valuable contribution and should be on the bookself of every medical student and practitioner.

WALTER SCHMITT

Modern Pharmacology

Pharmacology and Therapeutics. Originally written by Arthur R. Cushny, M.D. 13th Edition revised by Arthur Grollman, M.D. & Donald Slaughter, M.D. Philadelphia, Lea & Febiger [c. 1947]. 8vo. 868 pages, illustrated. Cloth, $8.50.

This book is therapeutically nihilistic to most of us old timers. Materia Medica is considered obsolete and Pharmacology no longer essential in medical curricula as the pharmacist is really "the man behind the drug gun." The physician merely "parrots the prescription."

The section on Toxicology is the most painstakingly masterful treatise as yet in print. It represents years of carefully checked research and scientific investigation.

THOMAS F. NEVINS

Chest Roentgenology

Principles in Roentgen Study of the Chest. By William Snow, M.D. Springfield, Ill., Charles C. Thomas, [c. 1946]. 4to. 414 pages, illustrated. Cloth, $10.00.

To the bedside clinician who has a familiarity with chest X-rays, this book is like a special P-G course.

Presentation is excellent and brief histories and physical findings add interest to the individual cases. Of particular value are the follow-up findings.

In brief, a wealth of valuable matter is splendidly presented.

FRANK BETHEL CROSS

Prescriptions

Manual of Prescription Writing and Medical Jurisprudence. By Harold N. Wright, Ph.D. 2nd Edition. Minneapolis, Burgess Publishing Co., [c. 1944]. 4to. 117 pages, illustrated. Fabricoid, $1.50.

This Manual consists of mimeographed sheets in loose-leaf form. It presents the mechanics of prescription writing in a clear and simple manner. Any physician called upon to prescribe drugs for his patients can glean much valuable information from this book.

CHARLES SOLOMON

Peritonitis

The Treatment of Peritonitis and Its Aftermath. By Thomas B. Noble, Jr., M.D. Indianapolis, A. V. Grindle, [c. 1945]. 8vo. 233 pages, illustrated.

The author attempts to clarify what he terms the Mechanics of Peritonitis and its treatment. The success of this effort must be left to the individual reader.

His conclusions are based on his experience in World War I and confirmed by later research.

STANLEY B. THOMAS

Hygiene for Students

Hygiene. A Textbook for College Students on Physical and Mental Health from Personal and Public Aspects. By Florence L. Meredith, M.D. 4th Edition. Philadelphia, Blakiston Co., [c. 1946]. 8vo. 838 pages, illustrated. Cloth, $4.00.

Now in its fourth edition this is like an entirely new work. It is well printed on excellent paper.

The material is presented in a clear concise manner so as to be easily understood by the high school student, and offers to the teacher a well arranged program for instruction.

C. T. GRAHAM-ROGERS

Proctology

Proctology for the General Practitioner. By Frederick C. Smith, M.D. 3rd Edition. Philadelphia, F. A. Davis Co., [c. 1945]. 8vo. 466 pages, illustrated. Cloth, $5.00.

Now in its third edition, this book is presented as a working guide for the general practitioner. The field is covered fully and the keynote is practicality.

The book is very easy to read both from the standpoint of diction and printing, and it is easily handled.

A. W. MARTIN MARINO

BOOKS RECEIVED

for review are promptly acknowledged in this column: we assume no other obligation in return for the courtesy of those sending us the same. In most cases, review notices will be promptly published shortly after acknowledgment of receipt has been made in this column.

Equinococosis Genito-Urinaria. By Dr. R. Hornu Liria & Dr. F. Romero Aguirre. Barcelona, J. M. Massó, [c. 1946]. 8vo. 179 pages, illustrated. Paper, 30 ptas. (Estudios Monograficos de Investigacion Medica)

La Tromboflebitis Asecotena del Miembro Superior. By P. Piulachs, A. Rodriguez Arias & R. Arandes. Barcelona, J. M. Massó, [c. 1946]. 8vo. 99 pages, illustrated. Paper, 25 ptas. (Estudios Monograficos de Investigacion Medica)

Trastornos Psiquicos en Traumatisados Craneales. By Dr. Vallejo Nagera & Dr. Escudero Valverde. Barcelona, J. M. Massó, [c. 1947]. 8vo. 159 pages. Paper, 30 ptas. (Estudios Monograficos de Investigacion Medica)

Tuberculina y Asma Tuberculoso. By Dr. Román Alemany Vall. Barcelona, J. M. Massó, [c. 1946]. 8vo. 133 pages, illustrated. Paper, 30 Ptas. (Estudios Monograficos de Investigacion Medica)

Psicosis Paludicas y Atebrinicas. Trastornos Psiquicos en el Paludismo Espontaneo, en el Terapeutico y en Los Tratamientos con Preparados Atebrinicos. By Prof. E. Guija Morales. Barcelona, J. M. Massó, [c. 1945]. 8vo. 173 pages, illustrated. Paper, 30 ptas. (Colleccion de Monografias de Investigaciones Médicas)

Estudio Etiopatogenico-Clinico de los Sindromes de Insuficiencia Medular Hemocitopoyetica (Agranulocitosis, Trombopania Maligna, Panmielotisis). By Prof. P. Farreras Valenti. Barcelona, J. M. Massó, [c. 1945]. 8vo. 67 pages, illustrated. Paper, 20 ptas. (Colleccion de Monografias de Investigaciones Médicas)

Rehabilitation Through Better Nutrition. University of Cincinnati Studies in Nutrition at the Hillman Hospital, Birmingham, Alabama. By Tom D. Spies, M.D. Philadelphia, W. B. Saunders Co., [c. 1947]. 8vo. 94 pages, illustrated. Cloth, $4.00.

Tomorrow's Food. The Coming Revolution in Nutrition. By James Rorty & N. Philip Norman, M.D. New York, Prentice-Hall, [c. 1947]. 12mo. 258 pages. Cloth, $3.50.

A Textbook of Pathology. By E. T. Bell, M.D. Contributors, B. J. Clawson, M.D. & J. S. McCartney, M.D. 6th Edition. Philadelphia, Lea & Febiger, [c. 1947]. 8vo. 910 pages, illustrated. Cloth, $10.00.

Gastritis. By Rudolf Schindler, M.D. New York, Grune & Stratton, [c. 1947]. 8vo. 462 pages, illustrated. Cloth, $8.75.